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BIOFEEDBACK TREATMENT FACTORS AND THE ALLEVIATION OF MIGRAINE HEADACHE

by

SCOTT MORGAN SELLICK

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE

OF DOCTOR OF PHILOSOPHY

in

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FALL, 1982

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BIOFEEDBACK TREATMENT FACTORS AND THE ALLEVIATION OF MIGRAINE HEADACHE

submitted by SCOTT MORGAN SELLICK

in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

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ABSTRACT

Migraine sufferers may turn to a host of possible treatments for the relief of their suffering. Traditional medical approaches often times become too detrimental in themselves and alternate approaches must be sought out. Biofeedback is one method that, in conjunction with relaxation training, has proven useful in the alleviation of many migraine and muscle contraction headaches. The present study included a four-week baseline, a possible twelve treatment sessions, and an eleven-month follow-up (N = 48). On the basis of pre-treatment physiological profile and subjects' recovery responsivity (following removal of a psychosocial stressor), subjects were determined to be either slow EMG recoverers, relative to ST, or the reverse. These two groups were then assigned to either EMG training or ST training. Subjects received twelve sessions unless they were able to prove skill acquisition without feedback instrumentation being available (weaned), at which point their training ended. Hourly and then daily headache activity ratings were completed by all subjects. While improvement was noted in all groups over time, only one between groups difference was significant. The weaned group (demonstrated skill even when instrumentation not available) experienced a significantly greater reduction on all headache activity measures, received fewer biofeedback training sessions, and practised home-relaxation less, than did the not-weaned group of subjects. Seventy-five percent of the weaned subjects (n = 20) were much improved (50+% reduction) while 39.3 percent of the not-weaned subjects (n = 28) were much improved. Implications for further research and clinical application of results are discussed.

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CHAPTER ONE

INTRODUCTION

Biofeedback has become an avenue for behaviour change with increasing promise as more and more individuals endeavour to find alternatives to the traditional health care strategies. Biofeedback places the responsibility for change, for improvement, in the hands of the sufferer and it is this invitation to take control of aspects of one's life that has particular appeal to many. Individuals quickly discover that they can not only observe changes in a variety of physical states but that they can learn to initiate those changes with and then without the aid of biofeedback instruments. What was once thought to be automatic and not able to be consciously controlled, has now been acknowledged as being within the repertoire of the individual.

Biofeedback is information about an individual's state and about on-going changes occurring within the individual. The present study undertook to investigate the nature of the learning possible through biofeedback treatment procedures in the alleviation of migraine headache suffering. As information is made available to the migraine sufferer and practise results in the ability to control general body states and specific physiological functions, the individual is able

to utilize that which had been acquired in the laboratory or clinic and alleviate undesired states throughout the day. This study was designed to assess the utility of first assessing individuals to determine unique physiological characteristics that might suggest that one treatment might be more advantageous than another (EMG or Skin Temperature) and, second, to attempt to train as many individuals as was reasonably possible to be able to self-regulate specific physiological states with and then, more importantly, without the aid of biofeedback instrumentation.

Once migraine sufferers had been determined to be either slow to recover on EMG measures (relative to Skin Temperature measures), or slow to recover on Skin Temperature (ST) measures (relative to EMG measures), and were then randomly assigned to EMG treatment or ST treatment, training sessions began. The training was designed to encourage learning of the specific skill to the degree that control of that physiological system could be demonstrated quite clearly without the aid of feedback instrumentation. Too often the assumption is made that once control has been demonstrated, while sitting in front of the instrumentation, sufficient learning has occurred to ensure therapeutic benefits. This assumption was not made in the present study. Rather, pre-set levels of criterion, levels of skill at which point it would be acknowledged that self-regulation was clearly within the control of the individual, were established and once that was accomplished the individual was encouraged to demonstrate the control without the aid of feedback instrumentation. Once that was done, treatment was terminated.

It is the contention of the writer that it is not the amount of time spent in biofeedback training that it is important, rather that it is necessary that there be proof of a certain degree of skill acquisition. One does not receive a pilot license for simply being able to cause the dials to move, control must be demonstrated in such a manner that the instructor is confident that the instruments are moving because the trainee has not only recognized that he can initiate change but that he can do so purposively, and be confident in the direction and the amount of the change. Control must be demonstrated before learning is assumed and sufficient ability is rewarded.

After having been assigned to one of four experimental groups, (slow EMG recovery responsive/EMG training; slow EMG recovery responsive/ST training; slow ST recovery responsive/EMG training; slow ST recovery responsive/ST training), participants received treatment until able to prove that a skill had been acquired or, until the twelfth session. A four-week pre-treatment baseline period separated the physiological assessment from the beginning of treatment. Once treatment was terminated, each participant's headache activity was monitored throughout the subsequent forty-eight weeks. The long-term benefits of having learned a skill and having proven that learning had occurred are the focus of this thesis.

CHAPTER TWO

REVIEW OF THE LITERATURE

This chapter reviews the literature of the migraine headache and the various forms of treatment available to the migraine sufferer. The chapter is organized into four sections, First the migraine headache is outlined as to its nature, its etiology, and its treatment. Second, the biofeedback literature is presented with particular attention to its application to the treatment of migraine headaches. Third, the rationale for the present investigation is given and, finally, the chapter concludes with a brief outline of the specific research questions.

The Migraine Headache

Description

Approximately forty-two million Americans consult a physician each year for the relief of headaches and one-third of those people are suffering from migraines (Diamond & Medina, 1981) Individuals

may experience their first migraine headache any time between the ages of five and thirty with approximately fifteen percent of people under the age of forty suffering from migraine headaches in one form or another (Campbell, 1981). Although seventy percent of the migraine population may have a history of migraines in their families, it is not known whether the transmitting gene is dominant or recessive, or whether other physiological factors that may be inherited (and predispose one to migraines) are responsible for this familial trend (Daimond & Medina, 1981; Schnarch & Hunter, 1980).

Adams, Feuerstein, and Fowler (1980) suggest that the suffering from migraine is as prevalent as the common cold, quoting figures such as twelve million migraineurs in the United States, compared to forty-two million muscle contraction headache sufferers. Danskin and Crow (1981) report that among the stress related diseases, migraine headache and mental and emotional problems are tied for second place (twenty million sufferers each), behind hypertension (thirty-four million). Sargent (1979), while head of the migraine project at the Menninger Foundation reported that estimates range from fifteen million to thirty-five million sufferers in the United States. The estimates vary but the numbers are over-whelming regardless of the quoted source.

What is a migraine headache? It needs to be recognized that while there are a number of different types of migraine headaches, there is also a range to which a migraine can affect an individual. Migraine headaches may be moderate, severe, or incapacitating, and may occur as frequently as every couple of days or as infrequently as once every two years. Some individuals may have only one migraine in their life-time while others seem to have them for a life-time.

The classic migraine is experienced by approximately ten percent of the migraine population. There are three stages: the aura, the headache, and the postheadache. The aura (or prodromal stage) is an episode of focal neurologic symptoms usually lasting for fifteen to thirty minutes. The focal neurologic symptoms include visual disturbances, numbness or weakness on one side of the body, transient aphasia, thickness of speech, and vertigo. The visual disturbances are blurred or cloudy vision with superimposed bright zigzag lines sometimes of various colours, usually in only one visual field. Some sufferers will experience unusual symptoms such as abnormally increased hunger, nervousness, or mood changes as early as a day before the onset of the migraine pain. Often the sufferer will not be aware of the aura symptoms while others around him or her will begin to notice subtle changes in behaviour (speech, gait, mood, etc.), and will be able to suggest to the migraineur that perhaps they should sit down and attempt to relax or take medication before the pain phase actually sets in.

The headache phase is frequently accompanied by nausea, photophobia, vomiting, diarrhea, vertigo, tremors, excessive respiration and chills. The pain may be limited to one side of the head initially but may spread to the other side as the headache persists. This phase may last for an hour or as long as seven days.

The postheadache phase may be relatively pain-free but tenderness of the skull often remains for some time. Physical exertion often results in the return of the throbbing head pain.

The common migraine, experienced by approximately eighty-five percent of the migraine population, is characterized by the same headache symptoms as the classic migraine but without the aura. If there is a vasoconstrictive phase, it is apparently not severe enough to produce an aura of focal neurologic deficits. The pain of the common migraine may be of longer duration than that of the classic migraine.

The cluster migraine is experienced by approximately four percent of the migraine population. There is no aura with this headache but the pain may occur two or three times during a day. Although it is a vascular headache, the cluster headache is considered to be a variant of migraine by most authorities and to be a very different type of dysfunction by some. It is accompanied by nasal stuffiness, tearing, and unilateral facial flushing. It is common for a cluster headache to last for parts of every day for two or three months and then for the sufferer to be pain-free for a year or two. Little is actually understood about this headache.

For the sake of completeness two final types of migraines need to be mentioned. Opthalmoplegic migraines are rare and appear during the early adult years. The pain is moderate and unilateral, and is usually accompanied by extraocular muscle palsy involving the third cranial nerve. The symptoms are similar to those of a carotid aneurysm. Hemiplegic migraines are also very rare and are characterized by a loss of the visual field to one side or the other and by possible aphasia and motor/sensory loss.

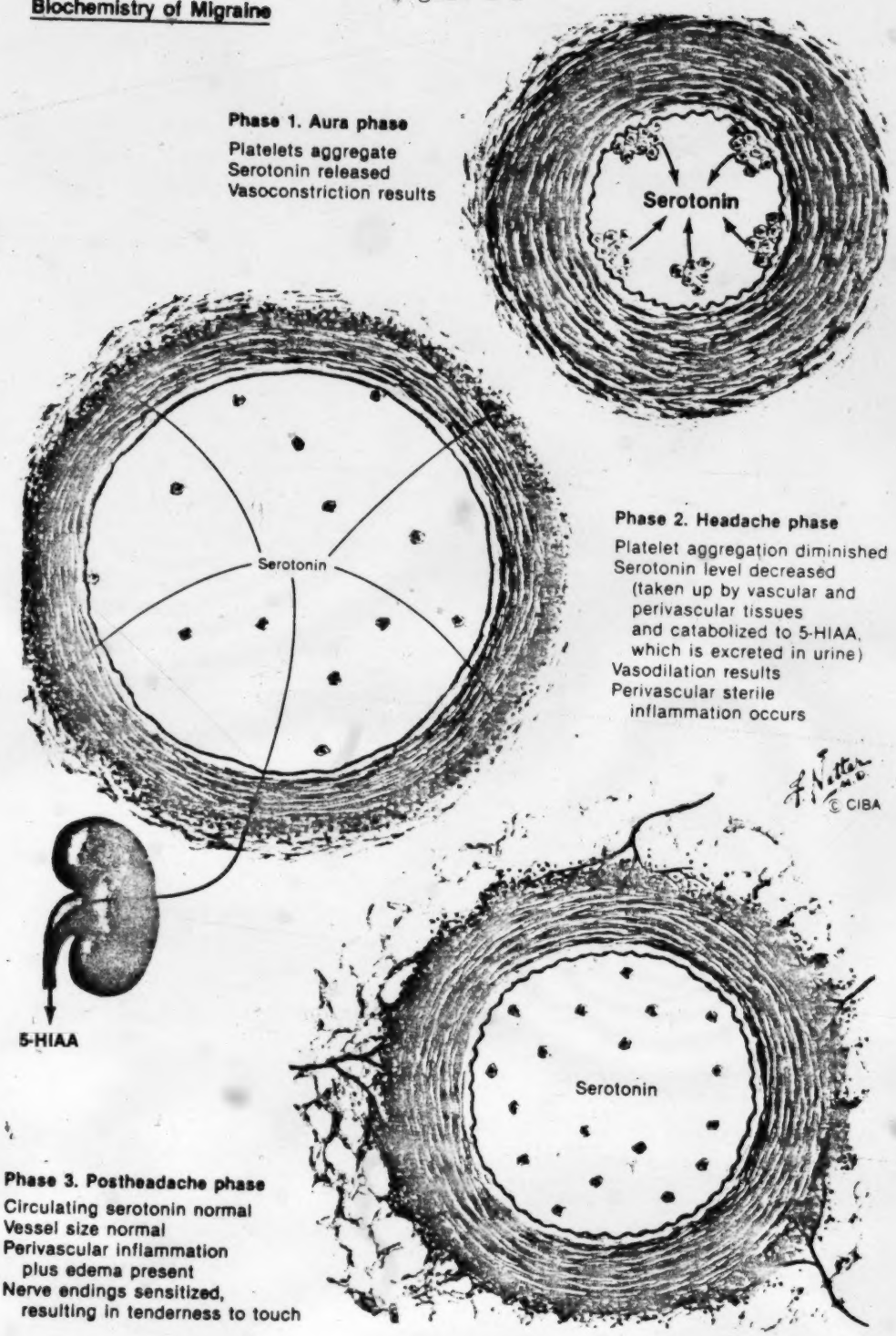
Pathogenesis

The vascular nature of migraine headaches (referring to classic and common migraines throughout the remainder of this paper) has been well established. However, many of the states and conditions necessary for a migraine to occur are not yet well understood. There are definite vascular changes, alterations in neural control, platelet changes, and the emergence of vasoactive substances but the complete picture has not yet been formulated.

The intracranial and the extracranial blood vessels are active throughout the migraine headache. There is the initial vasoconstriction (symptoms of the aura are evident at this point, if severe enough as in the classic migraine); vasodilation and sterile inflammation (headache pain); and a return to normal vascular states but with tenderness and edema (postheadache phase). Figure 2-1 presents the biochemistry of migraine.

The genesis of the vasocontriction is not clear although there is evidence of an inherent neurovascular instability in migraine patients (Diamond and Medina, 1981). There is a higher incidence

Figure 2-1



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(Diamond and Medina, 1981)

of disturbances in the autonomic system and of minor electroencephalographic abnormalities in migraine patients than in the rest of the population.

When a susceptible individual encounters a trigger for the migraine headache, both local and general changes take place. Locally, a predominantly unilateral cerebral vasoconstriction occurs that involves both the intracranial and the extracranial blood vessels. The innervated cerebral arteries begin to constrict as a result of neurogenic stimulation from stress or other factors, resulting in a reduced cerebral blood flow. Generally, the blood platelets begin to release serotonin. Once this serotonin is free within the blood, the result is further vasoconstriction of the innervated vascular system. If the result is vasoconstriction to a sufficient degree, an aura will develop.

This vasoconstriction causes local anoxia and acidosis (reduced alkalinity of the blood), and is soon followed by a reduction in serotonin levels as the serotonin is taken up by the vessel walls and perivascular tissues. It is at this point that noninnervated parenchymal arteries dilate. This is done in response to the state of anoxia and the sudden drop in blood serotonin. The sudden response of these noninnervated parenchymal arteries results in an increase in cerebral blood flow and local vasomotor changes in the innervated system of blood vessels, particularly in the ipsilateral extracranial and intracranial arteries. This sudden vasodilation accounts for the pain of the migraine. The pain is compounded by the fact that serotonin that was taken up by the

vessel walls sensitizes the pain receptors in the vessel walls and produces sterile inflammation around the vessels (Scheife and Hills, 1980). There are other theories but this is the most widely accepted.

The central dysnociception theory is one that deals with the brain's own pain system, suggesting that there is a dysfunction in the integration-modulation-inhibition of pain right at the brain stem level. Migraineurs seem to be deficient in 5-hydroxytryptamine (serotonin). When this is the case in an individual, there appears to a a disinhibition of central pain centres because serotonin is an inhibitory neurotransmitter for some brain stem neurons related to the perception of pain. Simply, if there is a lack of 5-hydroxytrptamine, more pain will be perceived. As blood vessels dilate as they might normally do but without pain, the individual deficient in this serotonin experiences intense pain (Scheife and Hills, 1980).

Verification of the changes in the blood serotonin throughout the phases of the migraine has come from Anthony and Lance (1967), who reported that a fall in plasma levels of serotonin occurred in eighty-five percent of the migraine attacks investigated. One would expect that serotonin levels would simultaneously increase in the urine as the kidneys excrete the free serotonin. This has been stated by Diamond and Medina (1981), who write that the serotonin is catabolized to 5-hydroxyindoleacetic acid which is excreted in urine.

Factors resulting in migraine headaches

Saper (1978) has provided a useful conceptualization of the migraine headache and its pathogenesis:

> Migraine can be viewed as a genetically determined physiological predisposition that is influenced by a wide variety of emotional, biological, and constitutional factors acting independently or in conjuction with each other and, additional unidentified elements, to precipitate an attack. (p. 2480)

Psychosocial stressors, physical stressors, and chemical stressors all manifest themselves in numerous and varying degrees. Some suffer from ulcers, some from hypertension, some from migraines.

Coffee, tea, and cola contain carbonic acid hydroxytryptamide complexes which are metabolized to serotonin. Chocolate, cheese, and food abstinence bring about sharp rises in plasma-free fatty acid levels which can also result in large quantities of serotonin being released into the blood. Rapid changes in hormone levels could be considered a stressor that some women are not able to cope with adequately. Many female migraineurs suffer two headaches each twenty-eight day cycle, one at time of ovulation and one just prior to menstruation. Rapid changes in blood glucose levels resulting from irregular eating habits and sleep pattern can result in migraine as well. Medical conditions may increase the frequency and severity of headaches (depression, hypertension), and medications for physical conditions can also result in migraines. Continued use or the sudden withdrawal from vasoconstrictive substances that contain perhaps

caffeine result in regular and sustained migraine headaches. Some medications containing vasodilating properties such as is used in the treatment of essential hypertension can also bring about headaches similar to migraine. Alcohol is a similar vasodilator and red wine in particular has been found to be a reliable trigger for some individuals. Approximately one quarter of all migraine sufferers report that foods of certain kinds initiate the attacks (Selby & Lance, 1960). Certain foods with a high content of tyramine for example, which initiates the release of serotonin into the blood, may trigger a migraine headache. Tyramine is found in alcoholic drinks, chocolate, nuts, citrus fruit, and aged cheese (Saper, 1978).

Physical exertion such as sudden exercise can initiate a migraine. Similarly, if physical activity is done too soon after a headache has ended, it may very quickly result in the return of the pain. Other physical stimuli such as bright sunlight, stuffy rooms, and changes in the weather may trigger migraines.

A final note needs to be made about the inheritability of migraine. Campbell (1981) reported that between fifty and eighty percent of patients have a directly homologous heredity with mother to daughter being the most common, followed by mother to son. There is also a close relationship between migraine and epilepsy. Campbell reported that the two conditions may be clinical manifestations of the same disorder. Epilepsy occurs more frequently in migraine patients; migraine occurs more frequently in epileptics.

The relationship between this genetic predisposition and

specific triggering factors will be discussed below as the diathesis-stress model is presented.

Treatment

Biochemical. The aim of biochemical treatment for migraine headache is to either alleviate an impending or existing headache or, prevent a headache before it begins. Anthony and Lance (1972) have classified drugs used to control migraine into four groups, those producing vasoconstriction (ergotamine tartrate), those stimulating the action of serotonin on receptor sites (Methysergide), those blocking beta-adrenergic receptors on blood vessels and therefore preventing vasodilation (propranolol), and monoamine oxidase inhibitors. Kudrow (1978) has suggested that even aspirin can be effective in that it inhibits platelet aggregation and the release of vasoactive agents which may be an important triggering event in the pathogenesis of migraine. More recent work in the review of the shunt theory of migraine has resulted in the notion that ergotamine (vasocontrictor) not only reduces carotid blood flow and thereby relieves the cranial vasculature of the painfully increased pressure, but that it exerts a direct action at the site of primary derangement, the cranial microcirculation. Spierings and Saxena (1980) further explain that this direct action is in the form of reversing the increased ratio of shunt to capillary flow, which, in turn, leads to improvement of tissue oxygenation. Interestingly, Spierings (1980) even questions the notion of the fall of the serotonin level as being responsible for the vasoconstriction. He stated that there is not even circumstantial evidence

available to support the assumption that serotonin, present in the plasma, exerts a tonic influence on the cephalic vascular bed.

Scheife and Hills (1980) provide a concise description of the pharmacological approaches to migraines, as have Diamond and Medina (1981). Before prescribing medication, three factors ought to be taken into consideration. First, the overall clinical evaluation of the patient and their symptoms needs to be completed to make the patient aware of how his or her headache is part of a lifestyle. This awareness of an individual's strengths and weaknesses, his usual responses to stressful situations, and his general attitude toward his headaches may provide addition clues as to how subsequent headaches might be reduced. Second, the nature and severity of the pain needs to be determined. Third, the individual must make a choice between treatment for individual acute attacks or for prophylactic therapy to reduce the frequency and severity of the attacks.

If abortive therapy is sought, the use of aspirin or acetaminophen may remedy the situation in mild cases. In more severe headaches, which is often the case in migraine attacks, ergotamine tartrate is usually the drug of choice. Ergotamine tartrate is a vasoconstrictor that is metabolized mainly in the liver and may be taken orally, sublingually, by inhalation, rectally, or parenterally. Intramuscular injection provides the fastest action, giving relief in about eighty-five percent of the patients; rectal administration and inhalation are effective in aborting about seventy percent of the attacks; an oral administration, while most convenient, aborts only fifty percent. If stomach nausea occurs, sublingual administration is advised.

There are problems using ergotamine tartrate however, since rebound headaches will occur if this compound is taken for two consecutive days. This means that patients who experience more than two migraines per week will not likely tolerate this drug, and for these cases there are other milder vasoconstrictors such as isometheptene. Unfortunately this drug is contraindicated in patients with high blood pressure. Dihydroergotamine, given intramuscularly, is also effective. The most effective of these compounds appears to be a combination of caffeine and ergotamine tartrate. The absorption of ergotamine is faster and more complete when given with caffeine, but the mechanism of the interaction is not clear. Individuals not advised to take ergotamine tartrate include those who are pregnant or have coronary heart disease, peripheral vascular disease, significant hepatic or renal dysfunction, anemia, Raynaud's phenonenon and thrombophlebitis.

When an acute attack persists for a number of days or perhaps a week or more, steriods will be administered. It is not uncommon for a small number of sufferers to be hospitalized during a prolonged attack and to be administered sedative-hypnotic agentsto enable them to sleep through the pain, or to be treated with narcotic analgesics.

For the patient whose headaches are occurring with increasing and unbearable frequency and for those unable to tolerate or do not respond to acute therapy prophylactic treatment needs to be considered. When attacks of migraine occur three or more times a month, prevention becomes the main goal of treatment. Methysergide maleate (Sansert) was the first prophylactic medication used with migraine.

Unfortunately, methysergide maleate must be taken on a regular basis for it to be effective (approximately seventy percent of patients respond favourably), and there is a strong potential for serious side effects. Diamond and Medina (1981) state that this drug can be used as a last resort in severe cases but should not be used longer than six months without a drug holiday of one or two months. Monthly checks are advised for heart or lung fibrosis and kidney problems, and a yearly intravenous pyelogram should be done as well.

According to Diamond and Medina (1981), the medication of choice in the prophylaxis of migraine is propranolol (Inderal), a beta blocker that inhibits vasoconstriction of blood vessels and blocks the aggregation effect of epinephrine on the platelets. Propranolol is not recommended in cases where asthma, chronic obstructive lung disease, congestive heart failure, or atrioventricular conduction disturbances are present.

Another medication used in the prophylaxis of migraines is cyproheptadine (Periactin), an antihistamine that blocks the histamine receptors and the serotonin receptors. Negative side effects such as drowsiness, impaired mental functioning, and weight-gain make this medication less desirable although it is good for children.

Ergotamine tartrate can be useful as a prophylactic medication but if too large a dose is taken over a number of days a rebound headache will occur.

Amitriptyline is a tricyclic antidepressant that has been found

useful in preventing migraines, especially those associated with muscle contraction headaches. Phenelzine sulfate, an antidepressant of the monoamine oxidase inhibitor variety also appears to have migraine prevention qualities. Again, the contraindications include hypertension, congestive heart failure, and liver disease.

Aspirin is effective in inhibiting platelet aggregation, as are sulfinpyrazone and dipyridamole.

Table 2-1 presents drugs, their dosage, and the side effects used in the abortive treatment of migraine headaches. Table 2-2 presents drugs, their dosage, and the side effects used in the preventive treatment of migraine headaches. It becomes apparent in reviewing the available literature that if a migraine can be aborted with aspirin or can be prevented with a low dose of propranolol the sufferer is perhaps very fortunate. The more severe migraine attacks need medication that is usually accompanied by unpleasant or even harmful side effects.
Adam, Feuerstein, and Fowler (1980), in their review of migraine headache literature concluded that 1) etiological factors remain unclear, 2) drugs are not adequate in relieving all suffering, 3) there are few well-controlled evaluations of treatment factors and, 4) biofeedback directed at modifying migraine pain appears to be promising.

Behavioural. Perhaps the most frequently used non-chemical treatment for migraine headaches has been biofeedback training where subjects have been taught to warm their hands. Blanchard and Ahles (1979), in their review of behavioural treatments noted

Table 2-1

Abortive Treatment

Type of Headache	Drug	Dosage	Side Effects
Migraine	Aspirin	600 mg every 4 hours p.r.n.	Most known side effects occur only with long-term administration of large doses
	Acetaminophen	600 mg every 4 hours p.r.n.	Similar to those of aspirin
	Ergotamine tartrate*	*Oral, sublingual:* 2 mg at onset of attack and 1 mg every 1/2 hour; maximum: 6 mg/day or 12 mg/week *Inhalation:* 1 dose every 4 minutes, up to 6 doses per day *Rectal:* 1-2 mg initially, repeated 1 hour later as needed, to maximum of 4 mg/day and 10 mg/week *IM or subcutaneous:* 0.5-1.5 mg, to maximum of 3 mg/week	Abdominal cramps, epigastric discomfort, diarrhea, nausea, vomiting, painful uterine contractions
	Dihydroergotamine mesylate	1 mg IM at onset and 1 mg every hour, to maximum of 3 mg/day and 5 mg/week	Similar to those of ergotamine tartrate
	Dexamethasone	16 mg IM, single dose; not to be repeated more than once every 3 weeks	Fluid and electrolyte disturbances, muscle weakness, GI disturbance, skin changes, cushingoid state; see package insert for others
	Isometheptene mucate†	130 mg at onset of attack and 65 mg every hour; maximum: 390 mg/day or 1,300 mg/week	Drowsiness, nausea, gastric disturbances
Cluster	Ergotamine tartrate*	Same as in migraine	See "Migraine"
	Dihydroergotamine mesylate	Same as in migraine	See "Migraine"
	Oxygen	By mask, 8-10 liters/minute for 5 minutes	None if taken as prescribed

*Drug of choice
†In combination with antiemetic

(Diamond & Medina, 1981, p. 16)

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Table 2-2

Preventive Treatment

Type of Headache	Drug	Dosage	Side Effects
Migraine	Propranolol*	*Oral:* 20 mg four times a day, increased in 2 weeks to 20 mg three times a day and 40 mg at night; maximum: 240 mg/day	Coronary ischemia, especially in patients with coronary heart disease, if drug withdrawn quickly
	Cyproheptadine†	4–12 mg/day	In adults: drowsiness, impaired mental performance, increase in appetite and weight gain
	Ergotamine tartrate	0.6–1.2 mg/day	See Table 1
	Amitriptyline	25 mg at bedtime; increased every 1 or 2 weeks until dosage reaches 100–200 mg/day	Dryness of mouth, mydriasis, blurred vision, constipation, bladder inhibition, dizziness, weight gain; see package insert for others
	Phenelzine sulfate	15 mg three times a day	Hypotension, insomnia, dryness of mouth, nausea, anorexia, constipation, dizziness, impotence, flushing, urinary retention, rash, red/green color blindness
	Clonidine	0.1 mg two to three times a day	Rebound hypertension if discontinued abruptly; constipation, drowsiness, disturbance of ejaculation, orthostatic hypotension, depression
	Methysergide	4–8 mg/day	See "Cluster"
Cluster	Methysergide*	4–8 mg/day	Fibrotic syndromes, nausea, vomiting, GI pain, diarrhea, drowsiness, dizziness, anxiety, hallucinations, severe psychotic reactions, muscle cramps, weight gain, hair loss
	Ergotamine tartrate	*Oral:* 1 mg three times a day for first week, then 1 mg twice a day for duration of cluster period	See Table 1
	Lithium carbonate	300 mg three times a day; maintain blood level between 0.5 and 1.5 mEq/liter	Tremor, nausea, urinary retention, polyuria, more rarely ataxia, exophthalmos, goiter, hypothyroidism
	Indomethacin	25–50 mg three times a day	Gastric disturbances, GI bleeding, corneal deposits; infections may be masked

(Diamond & Medina, 1981, p. 17)

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that a few controlled outcome studies have supported what many single group outcome studies have reported, that approximately sixty to eighty percent of patients improve on some measure of headache behaviour. Turin and Johnson (1976) reported that finger warming was effective on its own, without being accompanied by other behavioural approaches. Blanchard and Ahles point out however that the degree of improvement is not correlated with degree of temperature control achieved. The mechanisms involved remain to be investigated further.

Diamond, Diamond-Falk, and DeVeno (1978) concluded that temperature training (thermal) feedback, either in conjunction with autogenic training or alone, is beneficial in the treatment of vascular headaches. There is considerable difficulty in interpreting many studies for a number of reasons and it is the purpose of this study to investigate the treatment components of two different biofeedback modalities with migraine sufferers.

There is an interesting factor to the behavioural approaches that are used with any number of psychophysiological disorders. In contrast with the usual medical model approach, behavioural treatments tend to force the patient to look after himself for awhile. That is, special attention is often demanded on the part of the patient in order to assess the situation and arrive at a reasonable treatment plan. Mitchell and White (1977) examined the reactive effects of self-recording and self-monitoring on the frequency of migraine headaches in twelve sufferers over a period of sixty weeks, comparing their results with those of subjects involved in automated training

in behavioural self-management. Although self-recording and self-monitoring alone were not effective in reducing the frequency of migraine headaches, when combined with such practical skills as problem analysis, goal-setting, environmental planning and manipulation, muscle relaxation, mental relaxation, and self-desensitization, the reductions in headaches at time of three-month follow-up were fifty-five to eighty-three percent. Behavioural approaches to migraine headaches usually employ a number of strategies and ideally encourage general relaxation and home practise in addition to the clinic training of specific skills. The important factors are still be investigated.

Cognitive. Bakal, Demjen, and Kaganov (1980) reported positive results with forty-five individuals who had been diagnosed as suffering from migraine, muscle contraction, or combined migraine-muscle contraction headaches, using behavioural and cognitive techniques. Patients were taught how to modify the sensations, thoughts, and feelings associated with their headaches and how to intentionally control their particular pain-syndrome. The favourable effect was maintained at the six-month follow-up. This was very much an educational approach that was designed to make people aware of a number of variables associated with their headaches. This educational component (often taught indirectly through self-monitoring) appears to be more important than once thought.

Specific behavioural approaches using biofeedback instrumentation will be discussed at length in the following section.

Biofeedback with Migraine Headaches

Conceptual Framework

All men are not created equal. All men may follow the same route as they face psychosocial and psychophysiological stressors (Selye, 1977), but one must realize that there are vast individual differences in how each responds to stress. Some of us suffer from gastro-intestinal disorders, some from hypertension, some from cardio-vascular disorders, some from muscle contraction headaches, and some from vascular headaches. Each one of us responds differently to stressors, each one of us has our preferred "attempted solution" to life's stressors and it is often in the body's "attempted solution" that further problems arise.

Stress is a poorly defined concept considering it is something we all live with daily, all try to deal with, and all eventually succumb to in one way or another. Selye (1977) considers stress to be anything which causes an alteration of homeostatic processes. Burchfield (1979) suggests that Selye's definition is too limiting since it would follow that the constantly changing and moving human body is in continual stress. Burchfield defines stress as anything which causes an alteration of psychological homeostatic processes. A "stressor" is the specific stimulus in the transaction and the "stress response" is the organism's relatively nonspecific physiological response. Selye does provide a very workable definition of stress however, explaining rather well how we all must live with it and how our lives would not be as rich without it:

> You should not and cannot avoid stress, because to eliminate it completely would mean to destroy life itself. If you make no more demands on your body, you are dead . . . . A lash of the whip and a passionate kiss can be equally stressful! Although one causes distress and the other eustress, both make certain common demands, necessitating adaption to a change in our normal resting equilibrium . . . . Consequently, it would be unthinkable that anyone could, or would even want to, avoid stress. However, the more we learn about conditioning and about the ways to deal with the stress of life, the more we can enjoy eustress, which is the spice of our existence. (1977, p. 102)

Selye further explains that regardless of the nature of the stressor, whether it results in eustress or distress, one must be on guard against hyperstress and hypostress. In other words, it is not so much the nature of the stress that we should be concerned about as it is the amount (too much or too little) of stress that we deal with daily.

The world confronts each one of us with approximately the same amount of stress but each individual reacts differently to specific stressors and so, does not experience the same amount of distress. Some individuals will respond in such a way that the stressor results in eustress, the situation will be perceived to be pleasant. Others will respond such that the experience becomes distressing. Each person attempts to deal with the stressor

effectively but for the individual who experienced distress the attempted solution was less than adequate. Attempted solutions prove to be ineffective either because the environmental factors were overwhelming at a given time or because that individual's genetic predisposition was such that he was more susceptible to certain stressors.

Selye (1946) reported that chronically stressed rats had enlarged adrenal glands. More recently Sakellaris and Vernikos-Danellis (1975) reported that chronically stressed rats activated the pituitary-adrenocortical system faster than controls even though there was no difference of baseline values between groups. Selye has interpreted this type of finding as evidence of his General Adaptation Syndrome (alarm reaction, stage of resistance, stage of exhaustion), that each individual proceeds through in response to ongoing or intermittent stressors. Sakellaris and Vernikos-Danellis have interpreted the same results (although the stress conditions were different) as indicating that the animals were able to adapt, as opposed to being overcome with exhaustion. Perhaps Selye's third stage of the General Adaptation Syndrome is a stage of exhaustion for some and a stage of final adaptation for others.

The interesting finding in this animal literature is that the endocrine responses become conditioned to the cues for impending stress. In fact, organisms appear to respond to chronic intermittent stress before actual exposure to the situation. Fenz (1975) reported consistent results using human subjects. Measuring heart rate,

respiratory rate, and electrodermal response, comparisons were made between novice and experienced parachutists. Novice parachutists displayed high arousal on the morning of the jump and continued to increase their arousal up to the time of the jump. Experienced parachutists displayed normal levels early in the morning, reached a maximum of their arousal two hours previous to the jump, and were essentially at basal level at time of the jump. Their average arousal level was also lower than that of the novice parachutists. Both types of individuals were confronted with the same stressor but one type had learned to adapt and respond in a more efficient and effective manner. Comparisons were also made between those considered to be good parachutists and poor parachutists. These two groups approximated the experienced and the novice parachutists respectively, suggesting that experienced but poor parachutists have not learned to respond to the stress in any way different from the way in which they responded on their first jump. Good jumpers did learn new coping strategies.

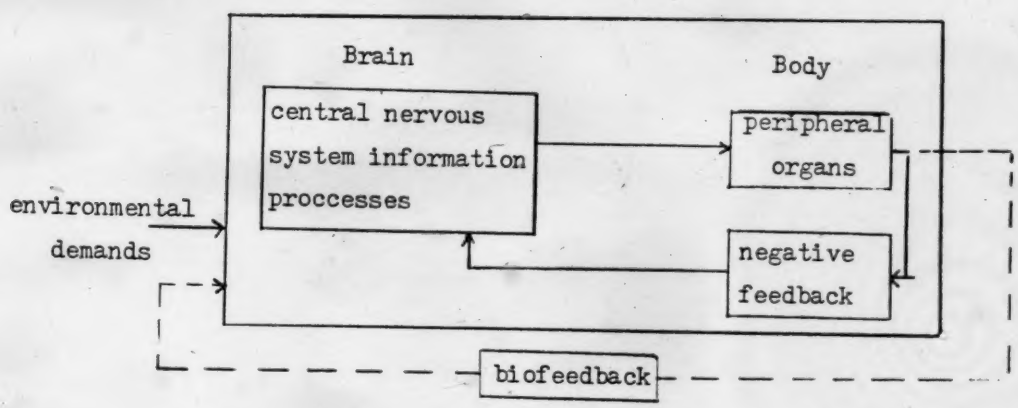
The human organism has a feedback mechanism within that provides ongoing information to the regulatory components of the body. As the environment changes various internal alterations become necessary in order to maintain homeostasis. The feedback system is necessary not only to maintain homeostasis but to conserve resources and to be effective in dealing with stress. Ideally the organism should be ready and waiting for the stressor and be in a relatively steady state having quickly recovered from the initial perceived stressor, as in the case of the experienced and the good parachutists. If the

corresponding feedback mechanism is not functioning adequately the organism will not be in a prepared stance nor be able to recover quickly, defend appropriately, or deal effectively with the stressor, as in the case of the novice parachutists.

Schwartz (1978) provides a useful conceptualization of the ongoing feedback mechanisms found within the organism. This depicts the complete process including the environmental stimuli, the central nervous system changes, the resulting changes in peripheral organ states, the negative feedback system informing the brain of the peripheral organ changes, and the role that artificial feedback can play in further enhancing the inherent feedback loop.

Figure 2-2

Environment, the Organism, and
Self-Regulation



(Schwartz, 1978)

This model presents a complete system that not only enables the individual to consider how one may influence the central and the peripheral nervous systems in times of relative health, but makes it possible to consider how one might remedy situations in times of disregulation and disease. While the inter-neuron communication within the human brain is carried on with rapid transmission via axons protected by myelin sheath, the unmyelinated fibres that return information to the conscious brain do so at approximately one-tenth the speed. The conscious brain does not associate the incoming information then with the environmental demands perceived, due to the lack of immediate and easily related feedback information.

Environmental demands often make it necessary for the brain to perform a regulatory function that inhibits peripheral organ activation. Should this peripheral organ change be maintained to the point where the organism experiences deterioration or injury, the negative feedback loop informs the brain of the detrimental effects, and the brain then makes necessary adjustments. It is this negative feedback loop itself that is often the source of pain, as in the case of stomach ache. When pain is experienced the individual may learn to change his behaviour and begin to eat more slowly, and make a mental note to not eat too quickly in the future.

This model is useful in that it illustrates the role of the central nervous system, the neuropsychological processes, and the feedback mechanism that regulates the organism in health and in disease. This model also accounts for the breakdown in the system

that often results in disease and distress that can no longer be coped with (Selye's exhaustion phase). Schwartz refers to this as the "instability disregulation" phase, the break-down in the healthy self-regulation. This break-down can occur at a number of different stages as the organism interacts with the environment, and for a number of different reasons. If environmental demands are over-whelming, the brain may be unable to attend to all the information being received; if the brain is disinclined because of genetic predisposition and subsequent social learning, the appropriate response may not be available; if the peripheral organ itself is a weak organ and is hypoactive or hyperactive to the neural stimulations, the negative feedback to the brain will not be accurate and the system will malfunction; if the feedback mechanism itself is not functioning properly, the information for self-regulation will not be correct or effective. Any one of these factors or any combination of them will result in a specific psychophysiological disorder.

The autonomic nervous system was once thought to be totally automatic, diffuse, and tightly coupled. We now know that this is a very simplistic view and that the autonomic nervous system has the capacity to learn specific self-regulatory actions and is in fact a sensitive system that not only learns to respond to various stressors but learns to anticipate those demands. The implication is that when an individual is provided with information about his physiological states and the ongoing internal changes, that organism is being taught something about a complex pattern of neural system

activity (Schwartz, 1977, p. 184), in addition to the information about a specific function at a given moment. Eastern methods have been employed for centuries to voluntarily control the actions of the autonomic nervous system, especially to induce parasympathetic reactions (Jencks, 1977). Only recently have Western schools of anatomy and physiology recognized that such changes can be initiated through deliberate volition.

Application of Biofeedback Instrumentation

Electromyographic training of the frontalis muscle has been recognized as a viable treatment approach with muscle contraction headaches for some time. EMG training has been effective in the treatment of some migraine headaches as well, but the favoured self-regulatory approach is skin temperature feedback training.

Green and Green (1979) suggest that, "Psychosomatic disease is, by definition, a medically undesirable physiological response to psychological stress. It is not in the head, but in the body, contrary to public opinion" (p. 157). What is being attempted then when an individual is asked to try and increase skin temperature in the hands with the desired effect being the loss of a migraine headache? Sargent, Walters, and Green (1973) suggest that the rationale behind hand-warming is a learning to "turn off" excessive sympathetic outflow. Sovak, Kunzek, Sternbach, and Dalessio (1977) reported evidence supporting a "hydraulic" model of migraine relief through hand-warming techniques. They reported that digital vasodilation was accompanied in four patients by a decrease in pulse volume, indicating vasoconstriction in the temporal and supraorbital arteries. The evidence is not conclusive and many question the shifting of

the concentration of blood from the head to the hands. Many investigators have proven that normal individuals can learn to self-regulate increases in hand temperature (Green & Green, 1977; Sargent, Green & Walter, 1973; Taub & Emurian, 1976), but Gainer (1978) has given excellent evidence that individuals can become quite accomplished at hand warming and yet are able to maintain their migraine headaches.

Hand-warming for the relief of migraine headaches was first suggested by Sargent, Green, and Walters (1972) after having conducted a physical relaxation study with a migraine sufferer:

> One of the subjects developed a migraine headache during both her laboratory sessions, brought on, she said, by fear that she would not succeed in warming her hands . . . . We asked the subject to run through another simple test routine . . . it was very relaxing . . . . While this was going on, I was in the instrument room, studying the physiological records as they emerged on the polygraph. Suddenly, at about the ninth minute, I noticed a rapid vasodilation in both hands, and a corresponding increase in hand temperature of about ten degrees Fahrenheit in the next two minutes. When the test was over I went into the experimental room and asked, "What happened to you a couple of minutes ago?" She replied, "How did you know my headache went away?" (Green & Green, 1977, p. 35).

In the single case reported by Gainer, a twenty-six year old migraine sufferer had been given assertiveness training and systematic desensitization before being introduced to temperature training. The temperature training phase consisted of ten sessions of training for a total of five hours of training. By the fourth session the woman was able to increase her skin temperature of her hand by eighteen degrees Fahrenheit. She was able to do this without the feedback instrumentation as well as with it. However, the migraines persisted and not until she had been taught how to discriminate the onset of a change in her hand temperature was she able to bring about the skin temperature increase and abort the headaches. Only by hand warming at specific times in response to specific temperature changes was this woman able to deal with her headaches.

There has been considerable discussion concerning the actual nature of cerebral blood flow in migraine headaches. Edmeands (1977) pointed out that ergotamine tartrate does not affect cerebral blood flow and yet it does abolish migraine headaches. Frequently an individual will have headache confined to one temple but there will be hyperperfusion on the entire ipsilateral hemisphere. It is also possible to have increased blood flow that is bilateral while the headache is strictly unilateral. The conclusion is that any theory that explains temperature training effectiveness in terms of shunting blood from head to hand and thereby reducing concentration and pressure in the head is, at best, incomplete and too simplistic.

Rather than resulting simply in a shift of blood concentration (which does occur but may not be the reason for the cessation of migraine pain), the temperature training brings about a rebalance of the entire vascular system. Taub (1977) considers the temperature training to bring about specific blood flow changes in some individuals while in others what is being observed is a general relaxation response.

Green and Green (1979) provide a more complex and yet logical account of the skin temperature training paradigm. Self-regulation of autonomic processes and, specifically, of skin temperature is a function of perception and imagination on the part of the feedback trainee. It is not the movement of the needle indicating temperature changes that results in the treatment effect but the imagining and visualization of what the needle is indicating to that person. The individual perceives a change and responds to that change emotionally and cognitively. As a result of the emotional and cognitive reactions there is a limbic response in the form of electrophysiological activity that is passed on to the hypothalamus and to the pituitary gland. The limbic signals are thereby translated by the hypothalamus and pituitary glands into autonomic and hormonal changes. The hypothalamus and pituitary glands are responsible for homeostatic balance but do not appear to be capable of initiating change in physiological states. Change is a result of limbic activity which responds to the mentioned emotional and cognitive reactions of the individual to the movements of the needle (indicating skin temperature changes). If additional information can be made available through

biofeedback instrumentation, the individual will be able to take that information and, utilizing various emotional and cognitive strategies, bring about the limbic responses that will result in the desired effect.

The original work of the Menninger Clinic (Sargent, Green, & Walters, 1972) have been subsequently reported by a number of authors (Adler, 1979; Diamond, Diamond-Falk, & DeVeno, 1978; Taub & Stroebel, 1978; Turin & Johnson, 1976; and Yates, 1980). Although the notion that hand warming brought about a shunting of blood away from the head was considered to be perhaps too simplistic, little had been offered as alternate explanations or as a more complete explanation until recently. Most explanations seemed to be limited to such phrases as "migraineurs have more reactive sympathetic nervous systems than non-migraineurs and hand warming brings about a calming effect on the autonomic nervous system" (Payne, 1979). Kewman (1978) concluded that because hand warming reduced migraine activity and hand cooling increased the migraine activity one might assume a direct relationship but that in itself, hand warming cannot account for the change. Apparently even those who had been unable to demonstrate that they had learned the hand warming skill were able to demonstrate headache activity improvement at times when those who did have the learned skill were unable to demonstrate headache activity improvement. Friar and Beatty (1976) compared the results of nine individuals trained in vasoconstriction of the extracranial arteries (measured from the surface of the skin with two pressure-transducing plethysmographs, recorded in pulse

wave amplitude), with the results of nine individuals trained in vasoconstriction of blood vessels in the hand. The nine individuals trained in vasoconstriction in the extracranial arteries not only demonstrated that skill but showed a marked improvement in headache symptomatology. Those trained in vasoconstriction at the hand-site demonstrated no change in the state of extracranial arteries or of headache activity. This study is suggesting specific vascular changes in the cranial area can account for changes in migraine activity. A more recent study by Bild and Adams (1980) compared the results of cephalic blood volume pulse (BVP) feedback, EMG feedback, and a waiting list control group in the modification of migraine headaches. While both treatment groups were superior to the control group in terms of decreased headache activity and medication intake, the BVP feedback group had results similar to the EMG feedback group. It appears that specific training to over-come pain is equal to the general therapeutic effect of EMG training for general relaxation, for the alleviation of pain with migraine sufferers.

Cohen, McArthur, and Rickles (1980) made similar comparisons among four groups: finger warming/forehead cooling, frontalis EMG relaxation, alpha enhancement, and vasoconstriction of the temporal scalp arteries. Although all groups succeeded at alleviating headaches experienced per week, there were no significant differences between groups. Cohen et al concluded that biofeedback, no matter what type, has a modest treatment value for migraine sufferers and that there is no relationship between physiological change and successful

headache outcome. "The biofeedback effect is nonspecific. The most viable explanations are a relaxation effect or a sense of mastery and control. Both hypotheses need to be tested so that progress will continue toward a maximally effective behavioral treatment of migraine headache" (p. 479).

A final study must be cited to point out that there is evidence of a specific vascular change component to temperature training that is an entity beyond simply being a general relaxation response. Claghorn, Mathew, Largen, and Meyer (1981) measured the cerebral blood flow in eleven migraine patients and nine normal volunteers. Half of each group had been assigned to either hand-warming feedback treatment or hand-cooling feedback treatment. Both normals and migraineurs displayed shifts in regional cerebral blood flow. The migraine group showed significant differences in the direction and/or magnitude of cerebral blood flow change but the normal group showed many dissimilar patterns. There appears to be strong evidence of a unique vasomotor response in migraineurs, a result of complex interaction of biochemical, neurogenic, myogenic, and metabolic systems. This means that the temperature training and the hand-warming in particular could be bringing about changes in the reticular activating system, in the sympathetic tonus of cerebral arteries, or in the metabolic states at the neuron level. While normal volunteers were as capable of learning the feedback techniques as were the migraineurs, the cerebral measures were quite different, suggesting that it is the way in which migraineurs respond to a stressor or stressors--not the

presence of a stressor--that is the important factor in the migraine etiology.

It appears then that the "hydraulic" model is an insufficient explanation for migraine headache alleviation through biofeedback for temperature training. A more likely explanation is that since finger temperature is a reasonably accurate guide to the emotional state of the individual; training that individual to increase finger temperature is an indirect way of training him to reduce arousal (Attfield & Peck, 1979). It is possible that when positive outcomes have not been forthcoming, it has perhaps been due to the fact that temperature training is an indirect way of training someone to alter autonomic nervous system activity, and that such an endeavour would naturally take more time and more expertise than one might assume following a few sessions of training (where general relaxation response activity would be evident and might be interpreted as being evidence of vascular control).

The use of electromyographic (EMG) feedback was first suggested as an effective way of inducing generalized deep muscle relaxation by Budzinski and Stoyva (1969). They positioned sensors over the frontalis muscle and implied that if this very difficult task of learning to relax this specific muscle was accomplished, then the whole body would become relaxed. Although some recent literature indicates that as a treatment for muscle contraction headaches EMG frontalis feedback is no more effective than progressive relaxation training (Cox, Freundlich, & Meyer, 1975; Haynes,

Griffin, Mooney, & Parise, 1975), the point is still debated. For example, Hutchins and Reinking (1976) reported that there was a significantly greater improvement in headache activity (muscle contraction) in subjects who were given EMG feedback and relaxation instructions, than was found among subjects who received only relaxation instructions.

Electromyographic training for headaches deals with sensing the amount of tension in the frontalis muscle, a muscle once considered to be an excellent point at which to focus in order to bring about general relaxation. Surwit and Keefe have pointed out, however, that:

> The choice of the frontalis as a site from which general body tension can be monitored is an assumption lacking empirical support, [and], the assumption that changes in frontalis EMG activity will produce corresponding changes in subjective or behavioral response systems has little empirical support in . . . research studies. (1978, pp. 782-783)

Voluntary control of this striated muscle was first reported by Budzinski, Stoyva, and Adler (1970) who successfully treated five muscle contraction headache sufferers with EMG feedback. The question has remained however, whether the beneficial treatment effect is a function of a specific physical alteration of a particular muscle or of a general relaxation response.

Muscle contraction headache sufferers and migraine headache sufferers have elevated frontalis muscle tension levels (Bakal & Kaganov, 1977). This suggests that if the desired effect is general relaxation that re-aligns the autonomic nervous system and alleviates

migraine pain, perhaps EMG training, temperature training, and relaxation training might be equally effective. Cohen, McArthur, and Rickles (1980) have addressed this question. Reinking and Kohl (1975) had addressed a similar comparative question earlier in relation to physiological and self-reported measures of relaxation. They reported that in terms of speed of learning and depth of relaxation the EMG groups were superior to the strictly relaxation group (Jacobson-Wolpe), while all were superior to the control group. Cohen et al. compared four groups (finger warming-forehead cooling, frontalis EMG relaxation, alpha enhancement, and vasoconstriction of the temporal arteries), and reported that all groups had a drop in the number of migraine headache days per week with no change in intensity, disability, or length of headache. If the desired outcome is general relaxation, EMG training is superior to Jacobsonian muscle relaxation, but the apparent advantage of EMG training does not appear to translate into a beneficial effectiveness for migraine sufferers.

Similar results have been reported by Cox, Freundlich, and Meyer (1975) with tension headache sufferers and by Cott, Goldman, Pavloski, and Fabich (1981). EMG feedback training was coupled with relaxation training and compared to relaxation training alone. Although the sample size is small (N = 8), the results are definitely in favour of a general relaxation response being responsible for positive treatment effects. At the time of the one-year follow-up both groups had similar and significant reductions in mean hours of headache pain per day, headache severity, and medication injestion (Cott et al., 1981, p. 558).

It is possible that while EMG training is at times no better than general relaxation training (Jacobson-Wolpe), in particular individuals EMG training may well be a superior treatment. This would be the case with individuals who have remarkably high EMG levels and who had difficulty returning EMG levels to a lower state. This approach may be well suited for some individuals and enable them to initiate a general relaxation response and bring about a re-aligning of the autonomic nervous system. It may be this re-alignment that produces positive results in migraine treatment.

Placebo Effects

Throughout the centuries, practictioners of the healing arts have been plagued and blessed by the positive effects of inert treatments. Most "unexpected" or "unexplainable" outcomes have usually been considered to be the "nonspecific treatment factors present but not accounted for". Kazdin (1979) considers the term "nonspecific" to be an unfortunate one for it implies a number of undesired conditions. Nonspecific factors have usually been considered unimportant and to be factors that may have contributed to therapeutic gain but have not been central to, or at least sufficient for, therapeutic change. "The bifurcation of independent variables into nonspecific and, by implication, specific treatment factors is arbitrary and unfortunate" (Kazdin, 1979, p. 847). Specific variables have usually been separated from nonspecific variables by something akin to intuition, the former somehow being more important than the latter. Empirical evidence should be the

criteria used to determine whether something is central to and sufficient for therapeutic change. What has often been the case is that anything that may have been part of a treatment program but which had not been, or could not be, tied into a theory was not given importance. Kazdin (1979) believes that "research often places a premium on making predictions about variables that not only produce change but also support a theoretical position about psychopathology or psychotherapeutic change" (p. 847).

Wilkins (1979) suggested that too many researchers have been interested in demonstrating how their treatments have been beneficial while being quite separate from nonspecific treatment factors. Therapy cannot however, be administered free and independent of nonspecific treatment effects. It does become important to question nonspecific effects if two treatments differ in terms of their credibility to the client and in treatment-generated expectancies. Researchers need to be careful and ensure that nonspecific effects are equally present in each treatment used and not so concerned as to whether nonspecific variables are present or not. Rather than attempt to control the nonspecific factors, researchers should be investigating them carefully and exploiting them to arrive at optimal and interpretable change. Kazdin (1979) suggests that the term "nonspecific" be dropped in favour of "common treatment factors".

Grunbaum (1981) has attempted to clarify the confusion that has often risen as a result of inconsistent usage of the placebo concept. "Throughout the . . . literature, the terminology used to characterize placebos is misleading, imprecise, and conducive to

conceptual confusion in research on their effects" (p. 157). One article that he found to be quite inconsistent used the sentence "placebo effects can be quite specific", in one paragraph but then uses the term "specific" as a synonym for "nonplacebo" in the next paragraph. Grunbaum becomes almost impatient with the variations in definitions of placebo--nonplacebo and specific effects--nonspecific effects:

> To this conceptually dissonant discourse I say: in the case of a placebo it is, of course, recognized that incidental treatment factors may be potently remedial for [a problem], although the characteristic ones by definition are not. And if some of the incidental constituents are thus therapeutic, then the actual specificity of their activity clearly does not depend on whether the pertinent therapeutic theory is able either to specify their particular identity or to afford understanding of their detailed mode of action. Hence if some of the incidental constitutents of [the treatment] are remedial but presently do elude the grasp of [the theory], the current inability of [the theory] to pick them out from the treatment process hardly lessens the object specificity of their identity, mode of action, or efficacy. A theory's current inability to spell out certain causal factors and to articulate their mode of action because of ignorance is surely not tantamount to their being themselves objectively 'non-specific' as to their identity over and above being unknown! (Grunbaum, 1981, p. 164)

The question of placebo effects surrounding biofeedback treatment has been addressed by a number of authors. The conclusion seems to be that there are psychological factors involved in biofeedback treatment that are above and beyond physiological or chemical explanation. These have unfortunately been given the classification of placebo or non-specific treatment effects. The theory in which biofeedback training is couched needs to account for these placebo effects so that they may be identified, understood, and included as specific treatment effects.

Blanchard, Andrasik, Ahles, Teders, and O'Keefe (1980) reviewed the migraine and muscle contraction literature. Their conclusion regarding migraine headaches was that temperature feedback alone, relaxation training alone, or temperature feedback combined with autogenic training were equally effective and were significantly superior to medication placebo. The average percentage improvement in migraine patients treated with one of the behavioural approaches ranged from fifty-one percent to sixty-five percent, while improvement due to medication placebo was approximately sixteen percent. They conclude that there must be a common feature among behavioural techniques that account for improvement in migraine headaches, that being relaxation. This interpretation casts doubt upon the theory that temperature feedback results in specific vascular changes directly. Sovak, Kunzel, Sternback, and Dalessio (1978, 1981) suggest that it is the reduction in sympathetic outflow that results in migraine relief.

Biofeedback lends itself easily to the work of Bandura (1977) and the concept of self-efficacy. His theory casts light on the aspects of biofeedback that have perhaps not been well-integrated

into existing theories of behaviour change. Bandura believes that if the appropriate skill neccessary to accomplish a task is within an individual's repertoire, "efficacy expectations" will be among the major determinants of the nature and degree of performance attainment. "Efficacy expectation is the conviction that one can successfully execute the behaviour required to produce desired outcomes" (Bandura, 1977, p. 193). The stronger the self-efficacy, the more active the efforts will be, the greater the likelihood of success.

There are four sources of efficacy expectations. First, performance accomplishments provide specific information about present abilities compared to past abilities, about how performance compares to that of another individual, and about how present strategies are affecting behaviour. Second, vicariously experiencing the accomplishments of others serve to boost one's personal expectations. Seeing another perform a threatening task without undue suffering can result in a change in personal beliefs about consequences of one's own effort. Third, verbal persuasion can also result in changes in personal beliefs about one's own ability. It is not, however, as effective as the previous two sources of efficacy expectations. Fourth, emotional arousal provides information about one's ability to perform. High arousal usually hinders performance and so individuals are likely to experience greater self-efficacy when they are not aroused in the face of a potentially threatening situation. Biofeedback, desensitization, relaxation training, and other behavioural manipulations of arousal can be extremely useful in developing an expectation of competency within the individual.

Migraine headache sufferers may have very low levels of

efficacy expectation. They may have been told that the literature indicates that if an individual is able to learn how to relax in certain and specific ways and bring about particular changes in cerebral vasculature or arrive at a state of general relaxation, migraine pain will be alleviated. However, migraineurs have usually suffered through years of discomfort and inability to over-come their syndrome; their self-efficacy is low. They may readily acknowledge that if they were able to master a technique they would no longer suffer as they do but, they may be slow to accept that they personally could acquire the necessary skill.

In order to increase the migraine sufferer's sense of self-efficacy and level of efficacy expectation any treatment package must attempt to provide the above-mentioned four sources of efficacy expectations. First, biofeedback does provide moment to moment reports on accomplishments and level of achievement. Second, where possible biofeedback training should be done in the company of another trainee so that vicarious learning may take place. Third, the therapist should be quick to encourage and to point out where certain strategies have proven to be useful. Fourth, where emotional arousal is high the migraine patient should be taught to recognize the state and to learn to anticipate such conditions before they reach unmanageable proportions. They should be taught how to recognize potentially high arousal situations and how to return to a more relaxed and controlled state (relaxation exercises in laboratory and at home, desensitization, biofeedback training). Each of these has been encorporated into the present

study's treatment packages with an endeavour to make each component of the treatment package equal across both EMG training and temperature training. This is not in an effort to control for nonspecific treatment effects or placebo factors but rather to maximize specific treatment effects (non-placebo factors) in each treatment equally.

Present Investigation

Silver, Blanchard, Williamson, Theobald, and Brown (1979) have reported on the initial results of their study comparing temperature biofeedback with autogenic training to progressive relaxation and to a waiting list control group. While progressive relaxation seemed to be a better treatment than temperature training after six weeks of treatment, at the time of the one, two, and three month follow-ups, no differences between groups were found. Headache measures included frequency, duration, intensity, and medication consumption. While both groups maintained their levels of success to the point of the one year follow-up, the groups did not differ from one another.

The present study investigates headache variables throughout a one year period from time of beginning treatment (EMG biofeedback and temperature biofeedback). Carney (1981) reported that EMG training and combined EMG training and temperature training were more effective in the reduction of migraine headaches with subjects who demonstrated (during a pre-treatment psychophysiological stress profile session) relatively slow temperature recovery compared to EMG recovery, than was temperature training alone. Temperature

training was most effective in reducing the headache behaviour of those subjects who had been predetermined as being slow EMG recoverers compared to temperature recovery. No other grouping of subjects resulted in any differential results.

The notion of differentiating subjects physiologically on the basis of the nature of recovery data is supported by previous research. Groups of individuals are most meaningfully differentiated physiologically on the basis of their recovery trends and not on their initial reactions to a stressor. Sellick, S. (1979) reported that a psychosocial stressor presented in a laboratory did not result in any initial stress reaction (heart rate change) that differentiated between high aerobic power (strong cardiovascular system) and low aerobic power (weak cardiovascular system). However, following the removal of the stressor, the two groups recovered at rates that were significantly different. This was consistent with earlier studies (Cox, Evans, & Jamieson, 1975; Sellick, M., 1977). Of particular interest was the finding that this physiological advantage was not reflected in any behavioural measures or in any of the self-reported measures of affect.

The present study divided subjects into two groups on the basis of individual recovery trends following the stressor, administered during the psychophysiological profile session. One group was determined to have had relatively long EMG recovery periods relative to a shorter temperature recovery period, and was termed EMG responsive. The second group was determined to have had relatively short EMG recovery periods relative to a longer temperature

recovery period, and was termed the temperature responsive group. Each group was then subdivided into two groups such that half of the EMG responsive group received EMG training while half received temperature training and, half of the temperature responsive group received EMG training while half received temperature training. Carney (1981) suggested that there was some evidence that EMG treatment was more effective in reducing migraine headache activity when used with temperature responsive subjects (longer temperature recovery relative to EMG recovery following stressor), than was the temperature treatment. Conversely, temperature treatment seemed to be more effective in reducing headache activity when used with the EMG responsive subjects (longer EMG recovery relative to temperature recovery following stressor), than was the EMG treatment. The present study organized migraine sufferers into the same types of groups.

Most migraine studies and treatment clinics suggest that headache sufferers attend a specific number of training sessions. The number varies from perhaps ten to twenty sessions in research settings and depends more perhaps on headache relief in clinical practice. Research has not looked at the notion of administering treatment until such time as expertise in the desired skill has been acquired and perhaps even proven not only with but also without the use of feedback instrumentation. EMG training and temperature training are both effective relaxation techniques but in terms of how much skill acquisition is sufficient to ensure that specific learning that will be beneficial to the migraine sufferer, little

has been done. This study was designed to answer the question concerning how much proven skill acquisition is sufficient for long-term migraine headache relief?

The reason that this question is important is that there may be a very logical explanation for previously reported findings where EMG training resulted in positive headache change in those who were relatively slow to recover from stress in terms of temperature recovery. Similarly, those who had received temperature training benefited most if they had been relatively quick to recover from stress in terms of temperature recovery. If individuals receive a pre-set number of training sessions it is very possible that during the treatment the most easily trained physiological system will be influenced first and to a greater degree. The effect of training in the most easily trained mode, that mode which recovered more quickly (the other mode being perhaps defective and therefore slower to recover), would be that the impaired mode would not be altered directly and, the therapeutic effect would be largely due to a general relaxation response.

Individuals who had been found to be quickest to recover in terms of EMG measures would likely benefit more from EMG training than would those found to be slow EMG recoverers. Temperature training would similarly be more beneficial with individuals who had been found to be quickest to recover in terms of skin temperature measures. An individual could perhaps be more easily trained in the more recovery responsive mode. Positive results were quickly realized but were perhaps superficial in terms of altering states essential for the

relief of migraine suffering.

The reasonable question is whether pushing subjects beyond the initial general relaxation response stage in biofeedback training to the point where they are able to initiate specific muscle and vascular changes would result in more positive changes in headache activity. It would be logical to assume that the individual who is slow to recover on EMG measures will benefit from temperature training or EMG training initially, as a general relaxation response occurs, but might benefit more from prolonged treatment to ensure that he acquires sufficient control over his impaired physiological recovery system, EMG. The individual who is slow to recover from a stressor in terms of skin temperature will benefit from prolonged temperature training to ensure that sufficient autonomic inhibition has been acquired.

The present study investigated this question of specific and sufficient control by setting levels of criterion, levels at which point it was agreed that a skill had been acquired. On the basis of manufacturers' recommendations and the reported temperature levels and changes observed in previous research (Taub & Stroebel, 1978), two criterion levels were set as targets for subjects to aim for and as levels to be attained and sustained both with and without feedback instrumentation. The EMG skill to be maintained (mean plus standard deviation over fifty-second phase) was 1.5 microvolts. The skin temperature skill to be demonstrated was bidirectional control of at least 2 degrees Fahrenheit in a limited period of time (three to five minutes). Surwit, Shapiro, and Feld (1976) reported that subjects were able to learn to consistently decrease hand

temperature by 4 degrees Fahrenheit and to increase hand temperature by .5 degrees Fahrenheit. However, Keefe (1975) reported that after twelve sessions subjects were able to change in either direction by about 1.5 to 2.0 degrees Fahrenheit. Taub (1977) reported that the mean change in hand temperature after three days of training (four fifteen-minute training periods per day) ranged from 2.2 degrees to 6.5 degrees Fahrenheit. Individuals within the top third of the subject pool who had been better able to control skin temperature were able to alter temperature in opposite directions during successive periods on the same day and routinely displayed ranges of eight to fifteen degrees Fahrenheit.

Sargent, Green, and Walters(1972, 1973) and Sargent, Walters, and Green (1973) first reported on finger temperature training with migraine patients but since then little has been added that might be of practical value to the practicing clinician. Yates (1980) concluded that recent studies have "provided no information to enable an evaluation of the success of peripheral finger temperature training, both because of confounding and the total failure to provide quantitative data of an appropriate type " (p. 239). Reading and Mohr (1976) have however provided specific information, reporting that temperature training resulted in an average reduction of seventy-six percent in the mean number of migraine headaches per week, and an eighty percent reduction in average hours of migraine headache duration at the time of the two-month follow-up. Temperature changes were in the 2.5 degree range (Fahrenheit).

Johnson and Turin (1975) investigated the utility of training to criterion in that they taught bi-directional control. Temperature

decrease training resulted in an increase in migraine headache activity while temperature increase training resulted in an decrease in migraine headache activity. Turin and Johnson (1976) further investigated this finding by having three subjects learn first to cool their hands before learning to warm them, and having four subjects learn just to warm their hands. All subjects learned to warm successfully and headache improvement was noted in all subjects while warming.

The present study was designed to investigate whether training to criterion would result in specific skill acquisition (as opposed to simply general relaxation) in a particular treatment modality that might result in migraine headache activity reduction beyond that normally brought about by non-criterion training. Subjects were seen for a maximum of twelve sessions over a period ranging from six to eight weeks. However, if criterion for skill acquisition was reached before the twelfth session, training was stopped. The individual was then required to prove that the skill had been in fact learned by producing the desired physiological changes without the aid of feedback instrumentation. When this was accomplished the subject was dismissed from the laboratory phase and was instructed in the follow-up procedures for monitoring headache activity throughout the subsequent months. Subjects who had reached criterion and subjects who had been weaned (proven skill without instrumentation) were expected to experience greater headache activity reduction than those subjects who may have learned how to relax but had not proven that they had learned a skill, and could control specific physiological

states.

Both EMG training and temperature training can result in generalized relaxation. If this is accomplished, it is believed to result in a shift from sympathetic to parasympathetic dominance in the autonmomic nervous system. Gellhorn (1967) and Gellhorn and Kiely (1972) proposed that reduction in muscle tension results in a change in the demands on the reticular formation and the hypothalamus which in turn brings about an increase in parasympathetic responding. This is the desired shift resulting from skin temperature training but it is possible that by ensuring specific skills have been learned, the sympathetic and parasympathetic nervous systems may be influenced more directly than through general relaxation training.

Research Questions

1. Is there a difference between training a migraineur on the modes that have been found to be slow or quick to recover following the removal of a psychosocial stressor?

2. Is there a benefit to training a migraineur to criterion that ~~is~~ reflected in a reduction in headache activity (with instrumentation available)?

3. Is there a benefit to training a migraineur to the point where competency can be demonstrated with and without the aid of instrumentation, that is reflected in a reduction in headache activity?

CHAPTER THREE

METHOD

Subjects

Subjects were obtained from throughout the city of Edmonton and the surrounding area, having been informed of the continuing research at the University of Alberta through a number of press releases. Although more than 100 people initially responded, sixty-eight were selected for inclusion in the study. Fourteen subjects failed to remain in the study through to the end of the first four weeks of follow-up, primarily due to conflicting summer plans, although perhaps the most correct explanation for half of the drop-outs might be lack of immediate-enough migraine relief. At the end of the one-year follow-up, forty-eight individuals were in regular contact with the researcher. It is these forty-eight individuals who will be discussed in the remainder of the paper. Inclusion criterion followed the guidelines as suggested by Adams, Feuerstein, and Fowler (1980), and were used during the initial telephone contact with each prospective subject (Appendix A). The subject population consisted of forty-four females and four males with ages ranging from twenty to fifty-nine.

Three group meetings were held on the university campus in order to briefly introduce the prospective subjects who had met the inclusion criterion to the recent use of biofeedback in the

relief of migraine headaches. Subjects who then agreed to participate were required to sign a treatment contract (Appendix B), and return with a physician's approval of their involvement in the research project (Appendix C). In addition, each subject was required to pay fifty dollars to cover expenses.

Research Design

On the basis of information obtained on each subject during a pre-treatment physiological profile session, subjects were placed in one of two groups. One group was comprised of twenty-four subjects who had been slower to recover according to EMG recording relative to temperature recovery following a three minute stressor (mental arithmetic task as part of the physiological profile session). This group was termed the EMG responsive group. The other group was comprised of twenty-four subjects who had been slower to recover according to temperature recording relative to their EMG recovery readings following the same three-minute stressor. This group was termed the temperature responsive group.

Within each of the two different responsivity groups, subjects were randomly assigned to one of two treatment groups. In other words, some subjects who were considered to be EMG responsive received EMG training while others received temperature training. The same random assignment to one of two treatment groups was carried out with the temperature responsive subjects. Over the course of the long-term follow-up the natural attrition was such that the four randomly assigned groups became unequal in terms of the number of subjects in each group. The final four groups would

be illustrated as follows:

GROUPS based on pre-treatment physiological profile	TREATMENTS randomly assigned	NO. of SUBJECTS in each GROUP	IDENTIFIER
Slow EMG Recovery Responsive	EMG Training	14	EMG/EMG
	Temperature Training	10	EMG/ST
Slow Temperature Recovery Responsive	EMG Training	11	ST/EMG
	Temperature Training	13	ST/ST

Once the four groups had been formed, treatment was ready to begin. Each subject was seen twice weekly along with one other subject. Each session lasted approximately forty-five minutes and was conducted by one of the three researchers. Each subject was seem by all three researchers throughout the treatment sessions, researcher assignment being carried out completely on a random basis depending upon time-table restrictions. A given subject was seen for a maximum of twelve treatment sessions. If criterion for successful mastery of the biofeedback skill was attained before twelve sessions, treatment was terminated. Otherwise, the subject would have received the twelve sessions. Criterion for successful mastery of the biofeedback skill was mentioned previously and will be outlined in greater depth in the following section.

Dependent Measures

Headache measures were made on an hourly basis during the one month of baseline data collection, during the treatment phase, and during the first month of post-treatment follow-up. During the subsequent months of follow-up, the headache measures were made on a daily basis. For the purpose of evaluating exactly what changes there may be have in different aspects of the headache behaviour a number of measures were collected. For the sake of meaningful data analysis and presentation of results, the headache measures throughout the complete research design were averaged across fourteen-day segments. Appendices D and E contain the headache record forms.

The first measure was number of days of headache during the fourteen-day time period. This measure would then be presented as a number ranging from zero to fourteen, and termed Headache Days.

The second measure was the number of hours of headache during the same time period. This measure would then be presented as a number ranging from zero to 336, and termed Headache Hours.

The third measure was an index that portrayed the average severity of the headaches that were experienced during the period. The daily 'average severity of headache' for each day of the fourteen-day period were summed. The average severity of headache would be a number ranging from zero to five; the Index (daily averages summed) would be represented by a number ranging from zero to seventy.

The fourth measure was the number of minutes spent deliberately endeavouring to bring about a state of relaxation of the nature introduced to each participant during the treatment phase. This

measure is presented as a number indicating the number of minutes spent doing the relaxation exercise during the fourteen-day period.

The number of weeks that individuals participated in the study ranged from fifty-six to sixty-four weeks. Due to the limitations of the recommended data analysis programs only data up to the end of the fifty-second week are being presented. In terms of actual follow-up information the time period is forty weeks, the first twelve weeks being the baseline period and the treatment period.

A comment on the validity of utilizing self-reported measures to substantiate real changes in headache behaviour is perhaps in order. Blanchard, Andrasik, Neff, Jurish, and O'Keefe (1981) compared the ratings obtained from significant others of treated headache patients with the daily headache ratings made by the patients themselves. The correlation between these two measures was significant ($r = 0.44$), suggesting that the results reported by the patients were supported by the reports of others in the patients' environments.

Apparatus

The treatment sessions for all subjects were held initially in a 20' X 30' room but external circumstances dictated that a move be made to a slightly smaller (8'X 20') but more suitable room. Two tables held all necessary biofeedback equipment for the two subjects, each of whom were seating in either a large lounge chair or a straight-backed chair (depending upon subject preference), facing the biofeedback equipment. Two filing cabinets, a table for the researcher to carry out data collection during the treatment session, and three table lamps for subdue lighting were also arranged within the laboratory. The pre-treatment physiological profile sessions had been completed in the larger room.

Electromyographic (EMG) levels were determined using Autogenics Systems Incorporated 1700 equipment. The manufacturer's recommendations for settings during treatment sessions were followed (a 100-200 Hz frequency bandpass, impedence levels less than 10,000 ohms, and a one second response averaging mode). Both visual (needle-meter) and auditory (clicking through headphones) feedback were available.

Temperature indications were provided using Autogenics Systems Incorporated 2000 equipment. The thermistor was secured to the finger-print area of the middle finger, non-dominant hand. Feedback was available in a visual or auditory mode, similar to that provided for EMG training. The auditory feedback (a variable tone through headphones) and the visual feedback (needle-meter with 0.1 degree Fahrenheit gradations) were available singly or in combination. This is consistent with previous research under similar circumstances (Carney, 1981).

EMG and temperature data were collected and recorded using Autogenics Systems Incorporated 5600 equipment with printer assembly.

During the four week pre-treatment baseline period and the treatment period, hourly headache data was collected using a progress chart (Appendix D). The same form was used during the first month of follow-up but due to the amount of time needed to conscientiously record data hourly and in the interest of maintaining a subject size of useful proportions, a daily progress chart was implemented (Appendix E). It was from these two charts that the information was gathered to create the above mentioned measures that were then recorded in terms of fourteen-day time periods for the purpose of analyses.

Procedure

Stress Profile

The pre-treatment physiological stress profile procedure was completed with each subject individually, the session lasting approximately forty-five minutes. The format was similar to that suggested by Stoyva (1979), and almost identical to that used by Carney (1981). Once the subject had received a brief explanation concerning the attachment of EMG electrodes and temperature thermistor, the flourescent lights were turned off leaving the room dimly lit by incandescent lamps, and the subject was asked to sit quietly with eyes open and relax for five minutes. EMG and temperature levels were being monitored during this and the subsequent fifteen minute period where the subject continued to relax but with eyes closed. The subject was then asked to silently perform a mental arithmetic task (to start at 1000 and continue to subtract sevens until told to stop), still with eyes closed. After three minutes the subject was asked to stop and to indicate how far he or she had subtracted. The final phase was then five minutes of resumed relaxation. It was the data obtained during this final five minutes of relaxation following the stressor that was used in the calculation of recovery responsivity and the assignment of subjects to either EMG or temperature response/recovery groups. Feedback was not provided during this session. (see Appendix F)

Physiological Recovery Responsivity Calculations

EMG recovery scores and temperature recovery scores were calculated based on the data obtained during the three minutes preceding the stressor, the three-minute stressor (mental arithmetic task), and the following five-minute period of relaxation.

The mean EMG level during the pre-stressor relaxation was calculated. It was then determined how many seconds it took for the EMG level to return to that point, following the end of the three-minute stressor.

The temperature data were dealt with differently. The initial drop in temperature at the time of the introduction of the stressor was calculated. It was then determined how many seconds it took for the subject to recover fifty percent of that initial temperature decrease.

T-scores were then calculated using the two recovery values obtained for each subject. These T-scores were then placed on a scattergram and a line was drawn in the fashion of a median split. In other words, a given subject's two T-scores resulted in a single point upon the scattergram and a line was then drawn dividing the scattered points into two groups of equal size. Subjects who were relatively slower to recover on EMG than on temperature when compared to other subjects comprised the EMG responsivity group. Subjects who were relatively slower to recover on temperature than on EMG when compared to other subjects comprised the temperature responsivity group.

Treatment Procedures

Following the pre-treatment physiological profile and a subsequent four-week period of baseline headache data collections, the two treatment procedures were implemented. The headache data collections was to continue throughout the treatment phase and for four weeks following the end of treatment. This included the hourly rating of migraine headache intensity as well as the time and duration of the relaxation exercises that each participant had been instructed in and encouraged to do daily.

The writer shared the responsibility for the administering of treatment with two masters students. All three researchers were familiar with the biofeedback treatment and were completing requirements for degrees in the counselling area with the Educational Psychology Department.

Participants during the study were seen twice weekly as consistently as individual schedules would allow. Subjects were seen in pairs as often as was possible although there was considerable variation in this procedural condition. Twelve treatment sessions, each lasting forty-five to fifty minutes, were available to each subject although if criterion for skill-acquisition was reached and the subject was able to successfully prove competence without the aid of the feedback, treatment was terminated. Since these procedures differed for the two treatment groups they will be explained separately below.

Throughout the weeks of treatment the subjects were instructed to listen daily to a thirty-minute audio tape. Specific instructions were given to each subject as to the use of the tapes and their importance.

EMG Training

The EMG biofeedback training began with the subject reading a short paper entitled, EMG Training Rationale (see Appendix G). This provided some information regarding how each subject might begin learning to control the feedback information and thereby bring about the specific physiological changes desired. The subject was connected to both EMG and temperature biofeedback equipment although temperature feedback was not provided at any time, only recorded. A given treatment session had four phases, the fourth phase being the only one during which feedback was available to the client. The subject was required to sit quietly and relax for five minutes with eyes open. Then the subject was to continue relaxing for an additional two minutes while baseline data were collected. The third phase was a built-in check for skill acquisition without biofeedback information being available to the subject. The subject was asked to decrease muscle tension for two minutes. A verbal report was provided by the researcher to inform the subject as to the success of the muscle tension reduction phase. This was of little value during the first few treatment sessions but provided valuable information for each subject to ascertain how their skill acquisition was being applied to situations where feedback was not available. It was at this point, during the final phase, that biofeedback was provided. Three five-minute periods of practice separated by two one-minute rest periods made-up this training phase. Visual and auditory biofeedback was provided with the instructions to decrease the muscle

tension in the head region.

The EMG training subjects were asked to take a few minutes to write down what strategies had proven to be useful in bringing about the desired changes in frontalis muscles relaxation, and to mention the feelings and sensations that had accompanied those changes. Appendix H contains the detailed and verbatim account of the EMG training procedure.

Throughout the weeks of treatment the subjects were instructed to listen daily to a thirty minute audio tape of progressive deep-muscle relaxation in the tradition of Paul (1966). A tape was made specifically for this subject group. The subjects were also strongly encouraged to make use of the relaxation exercises throughout the follow-up period as a way in which each person could maintain the level of control that had been learned using the biofeedback instrumentation.

Temperature Training

The temperature biofeedback training began with the subject reading a short paper entitled, Temperature Training Rationale (Appendix I). The subject was then connected to both temperature and EMG biofeedback equipment although EMG feedback was not provided at any time, only recorded. This treatment followed the same procedure as outlined above with EMG training, although the content did differ where necessary. Again, a given treatment session had four phases, the fourth phases being the only one during which feedback was available to the subject. Phase one consisted of having the subject sit quietly with eyes open, relaxing for five minutes. Phase two consisted of having the subject continue to relax while baseline data were collected for two minutes. Phase three was a two-minute segment where the subject was asked to increase skin temperature spontaneously, while not receiving any feedback information. A verbal report was provided by the researcher to inform the subject as to the success of the temperature-increasing phase under the no-feedback condition. Again, this proved to be helpful in that the subjects were able to discover how well they could induce temperature increase without the on-going biofeedback. Biofeedback was provided during the fourth phase but the format was slightly different here than in the EMG training. Three five-minute training periods were separated by to one-minute rest periods. If, at the end of the third phase, the subjects' skin temperature was below ninety degrees, their three sessions were as follows: increase skin temperature for five minutes, decrease skin

temperature for five minutes, and then increase skin temperature for five minutes. No-one was asked to decrease skin temperature until their finger temperature was higher than ninety degrees. If, at the end of the third phase of the treatment session the subject's skin temperature was above ninety degrees, the first five-minute training session began with five minutes of decreasing skin temperature. For such an individual session two would be five minutes of increasing skin temperature followed by the last five minutes of decreasing skin temperature.

The temperature training subjects were asked to take a few minutes to write down what strategies had proven to be useful in bringing about the desired temperature changes, and to mention the feelings and sensations that had accompanied those changes. Appendix J contains the detailed and verbatim account of the temperature training procedure.

Similar to the EMG training group, the temperature training group was instructed to listen daily to a thirty minute audio tape of progressive relaxation (autogenic, using imagery) focusing on the warming of the body and specifically of the hands. This tape was prepared specifically for this particular subject group. The subjects were strongly encouraged to make use of the relaxation exercises throughout a follow-up period as well, as a way in which each person could help maintain the level of control that had been learned while working with the biofeedback instruments.

In addition, these subjects were given a temperature sensitive band to wear around their finger. This band would give information

about their finger temperature at any given time and would therefore provide on-going feedback. Subjects were encouraged to wear the band for a few minutes before their daily relaxation exercises and then to compare the readings with those obtained during and after the relaxation period. Biotic band information is found in Appendix K.

Criterion Level for Skill Acquisition

One of the main components of this research endeavour that will have useful application for the clinician using biofeedback is the somewhat arbitrary setting of a criterion level of skill acquisition. The setting of a level of control that the particpant must demonstrate presupposes that the amount of time spent before a biofeedback instrument is not as important as is the question, 'Does the individual have a skill now that he did not have before?' Rather than having each subject attend a set number of training sessions, attention was given to the amount of control each person demonstrated from one treatment session to the next. Although no subject was seen for more than twelve sessions many were seen for considerably fewer sessions. The criterion for skill acquisition was necessarily different for each of the two treatment modalities. Subjects who were able to prove that they have learned a skill by meeting the level as pre-determined by the researchers ended treatment at that point. Once a certain level of competence had been attained using the feedback instrumentation the individual was required to bring about the same evidence of control without the benefit of the

instrumentation. This has a potentially powerful treatment component for the participant not only believes that he has received help but he also knows that he has learned a skill, that he has control over a particular aspect of his physiology.

For the subjects receiving EMG training they were required to maintain levels below 1.5 microvolts for each of five consecutive sixty second averages, for two consecutive five-minute training periods. An EMG reading was taken every sixty seconds, that reading having been averaged over the previous fifty seconds. The subject was expected to maintain levels below 1.5 microvolts for five consecutive one-minute sessions and then after a one minute break, repeat the skill for a second five-minute time period. Once this had been accomplished the subject was expected to demonstrate the same ability without viewing any feedback instrumentation. The participant proceeded through the five-minute relaxation periods alternately receiving feedback and then no-feedback. This format was continued until such time as the subject was able to maintain the EMG levels below 1.5 microvolts for two consecutive series of, five minutes with feedback and five minutes without feedback. When this had been accomplished, treatment was discontinued and the follow-up data collection time began.

For subjects receiving temperature training the researchers expected them to be able to increase skin temperature two degrees, then decrease skin temperature two degrees, and then increase skin temperature two degrees (or, decrease, increase, decrease). Once

that had been demonstrated with the use of feedback instrumentation, the subject was expected to again warm for five minutes with feedback, and then cool for five minutes with feedback. If that was done successfully (a change of at least two degrees each time), the procedure was repeated without the use of feedback instruments. If the participant was able to again warm two degrees and then cool two degrees within the five minute trials, and do that without feedback instrumentation being available, a skill was deemed to have been acquired. At this point treatment was discontinued and the follow-up data collection time began.

Weaning procedures for both EMG and ST training groups are provided in detail in Table 3-1.

Follow-up Format

Each participant in the research project who completed the treatment phase was contacted regularly throughout the subsequent follow-up period. Every three months a packet was mailed to each participant asking that data collected up to that date be mailed to the researcher using the envelope provided. Additional blank data sheets were included in the packet and the individuals were encouraged to continue recording their headache behaviour daily. Within a week or two of the packets being mailed the researcher made telephone contact with each participant to ensure that the information had been received and to make any clarifications that were necessary. The information that was mailed throughout the follow-up phase is contained in Appendix L.

Table 3-1

CRITERION FOR BEGINNING WEANING

1. EMG: The client is ready to begin weaning procedure when:

   the client has been able to maintain EMG levels below 1.5 microvolts (mean plus the standard deviation for that fifty-second segment) at each of the five readings for two consecutive five-minute practice sessions.

2. Temperature: The client is ready to begin weaning procedure when:

   the client has been able to warm skin temperature by two degrees (Fahrenheit), then cool by two degrees, and then warm by two degrees (or, cool, warm, cool), during three consecutive five-minute practice sessions.

FORMAT FOR WEANING

1. EMG: The client will follow the following procedure and treatment will be discontinued when it is successfully adhered to:

   the client will attempt to maintain EMG readings below 1.5 microvolts for five minutes while receiving biofeedback and then for five minutes while not receiving biofeedback. This will then be repeated once again (with, without; with, without).

2. Temperature: The client will follow the following procedure and treatment will be discontinued when it is successfully adhered to:

the client will attempt to bring about an increase in skin temperature (two degrees Fahrenheit) in five minutes while receiving biofeedback and then to decrease skin temperature by the same amount in five minutes while still receiving biofeedback. Then, the same procedure will be repeated without biofeedback. If changes occur with biofeedback and then without biofeedback, treatment and weaning are completed.

Six individuals were dropped from the study during the follow-up phase. Four individuals failed to return the data forms although they had been contacted repeatedly, one individual left the country and was not contacted, and one individual was not included due to the fact that her headache had been diagnosed as a cluster headache by her physician in the interim.

Characteristics of the study population (N = 48) are presented in Appendix M.

Practical Importance of Study

While ongoing research projects continue to sort out various components of biofeedback and determine the actual physiological mechanisms associated with various types of training techniques, the clinician desperately wants to know how best to meet the needs of the suffering migraineur. Barlow (1981) has written of the scientist-practitioner split, about how factorial designs with large numbers of homogeneous groups often say very little to the practitioner about clinical improvement in the individual subject. The notion of patient uniformity is a myth and if the goal of the clinician is to get the patient better quickly, the subjects need to be treated as individuals, not as large groups of near-identical subjects. This is to say that while the present study is a factorial design, little was done to ensure that subjects would not alter their lifestyle throughout the follow-up months, while much was done to facilitate the development of each subject's sense of self-efficacy, efficacy expectations, and outcome expectations.

Having assigned subjects to groups, every effort was made to make the treatment beneficial to each subject. Each was entitled to equal time in the laboratory unless criterion had been reached and subject successfully weaned from the instrumentation. Each was encouraged to practice for thirty minutes each day the relaxation skill being suggested by the biofeedback training. Audio tapes were available for this purpose (progressive muscle relaxation for EMG trainers, autosuggestive-imagery relaxation for temperature trainers). Each was asked to write down the strategies that had proven to be

helpful during the preceeding training session. Finally, the subjects were exposed to short periods of training with brief rest intervals so that fatigue might be avoided. EMG training consisted of three five minute sessions with a one-minute rest between sessions, each visit. Temperature training consisted of three five-minute sessions with a one-minute rest between sessions, each visit, and subjects would be required to alternately warm and then cool their hand temperature. Pre-training procedures held at the beginning of each treatment visit were consistent for all subjects.

CHAPTER FOUR

RESULTS

Data were analysed using a series of analyses of variance to assess the differences between groups from pre-treatment through to the end of post-treatment follow-up, in terms of headache activity variables. Multivariate profile analyses were used to assess differences between group profiles throughout the post-treatment follow-up, in terms of headache activity variables.

A number of different subject groupings were completed in order to answer specific research questions as outlined in Chapter Two.

EMG and Skin Temperature Recovery Responsivity Groups

Subjects were grouped according to whether they had been found to recover more slowly following the removal of a stressor in terms of EMG measures (relative to ST measures) or, in terms of ST measures (relative to EMG measures). This physiological assessment had been completed four weeks prior to treatment and was identical to the format as outlined by Carney (1981). Carney reported that by dividing subjects according to physiological recovery characteristics and then providing half of each group with EMG or ST feedback training, differential treatment effects were evident. EMG treatment was more effective with slow ST

recoverers; ST treatment was more effective with slow EMG recoverers. Subjects experienced greater migraine improvement when they received the training which corresponded to their physiological system determined to have been quicker to recover. Carney (1981) speculated that the quicker system was perhaps easier to bring under conscious control. It is plausible that an overactive sympathetic nervous system (responsible for blood-vessel constriction) is dysfunctioning due to inhibition of appropriate countering factors and that training to inhibit the inhibitory factor or, simply inhibiting the overactive SNS directly, will result in a stabilization.

EMG and ST recovery were expressed in seconds and were converted to T-Scores. After having placed each subject's point on the scattergram (Figure 4-1), a median split was executed such that half of the subjects became members of the EMG recovery responsive group (slow EMG recovery relative to ST recovery) and half became members of the ST recovery group (slow ST recovery relative to EMG recovery).

Members of each group were then assigned to either EMG training or ST training using a randomized block design procedure.

Analyses of Variance

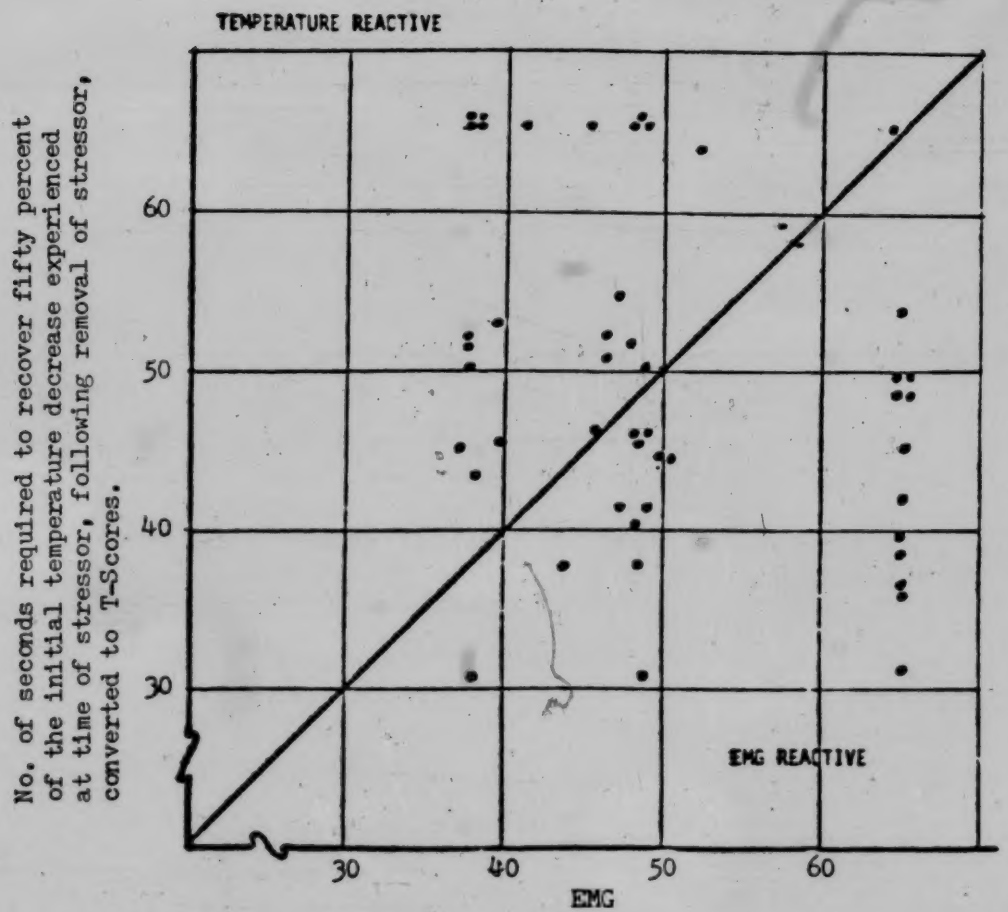
Six different series of Analyses of Variance were computed and will be presented below. In each case Factor B consisted of five repeated measures as outlined:

P-1	pretreatment	(four weeks)
P-2	treatment	(four to eight weeks)
P-3	posttreatment	(weeks 1 2 3 4)
P-4	posttreatment	(weeks 23 24 25 26)
P-5	posttreatment	(weeks 45 46 47 48)

Figure 4-1

Scattergram of EMG and Temperature T-Scores:

Derived from Pre-Treatment Stress Profile Data



No. of seconds required to return to pre-stress EMG levels, following removal of stressor, converted to T-Scores.

The headache activity variables monitored at each measure of Factor B were as outlined:

number of headache days per fourteen days

number of headache hours per fourteen days

summed average daily headache intensity per fourteen days

Experimental Treatment Groups Analyses of Variance

The headache activity variables were analysed using three two-factor Analyses of Variance with four levels of Factor A and five repeated measures on Factor B. The groups in Factor A were:

slow EMG recoverers/EMG training (n=14)

slow EMG recoverers/ST training (n=10)

slow ST recoverers/EMG training (n=11)

slow ST recoverers/ST training (n=13)

A significant period effect was noted when the four groups were collapsed. There was a significant reduction in the number of headache days, $F(4,176) = 21.92$, $p < .001$; in the number of headache hours, $F(4,176) = 16.33$, $p < .001$; and in the summed daily intensity index, $F(4,176) = 4.32$, $p < .01$. The cell means for each treatment group across five measures are presented in Table 4-1 (headache days, headache hours, headache index). The corresponding Analyses of Variance source tables are in Appendix N. No experimental group differences were noted.

The changes in headache activity from P-1 to P-5 are presented in Figure 4-2. Post-hoc multiple comparisons using the studentized ranged, Newman-Keuls method indicated that when the four treatment

Table 4-1

Headache Activity Variables

Four Treatment Groups'--Cell Means

Across Five Repeated Measures

		P-1***	P-2	P-3	P-4	P-5
Headache Days						
EMG/EMG*	n=14	5.500**	4.286	3.964	2.929	2.964
EMG/ST	n=10	8.200	6.800	6.900	4.650	4.650
ST/EMG	n=11	9.182	7.909	6.682	4.682	4.773
ST/ST	n=13	5.923	5.000	4.500	3.077	2.500
Headache Hours						
EMG/EMG*	n=14	58.571	52.714	43.250	30.821	28.893
EMG/ST	n=10	105.900	100.300	106.400	64.800	64.200
ST/EMG	n=11	91.091	71.455	54.545	30.182	34.455
ST/ST	n=13	58.385	55.308	43.308	25.423	20.231
Headache Index						
EMG/EMG*	n=14	10.429	7.714	7.714	6.821	6.643
EMG/ST	n=10	16.300	15.200	13.250	13.750	15.150
ST/EMG	n=11	15.455	12.000	10.409	9.909	8.773
ST/ST	n=13	11.231	9.692	7.385	7.077	6.808

*recovery responsivity group/biofeedback training group

**values are the averages of two fourteen-day periods

***P-1 (pre-treatment) P-2 (treatment) P-3 (posttreatment #1) P-4 (posttreatment #2) P-5 (posttreatment #3)

Figure 4-2

Headache Activity Variable

Four Treatment Groups'--Cell Means

Across Five Repeated Measures

EMG recovery responsive/EMG training · · · · ·
EMG recovery responsive/ST training – – – – –
ST recovery responsive/EMG training ————
ST recovery responsive/ST training – · – · –

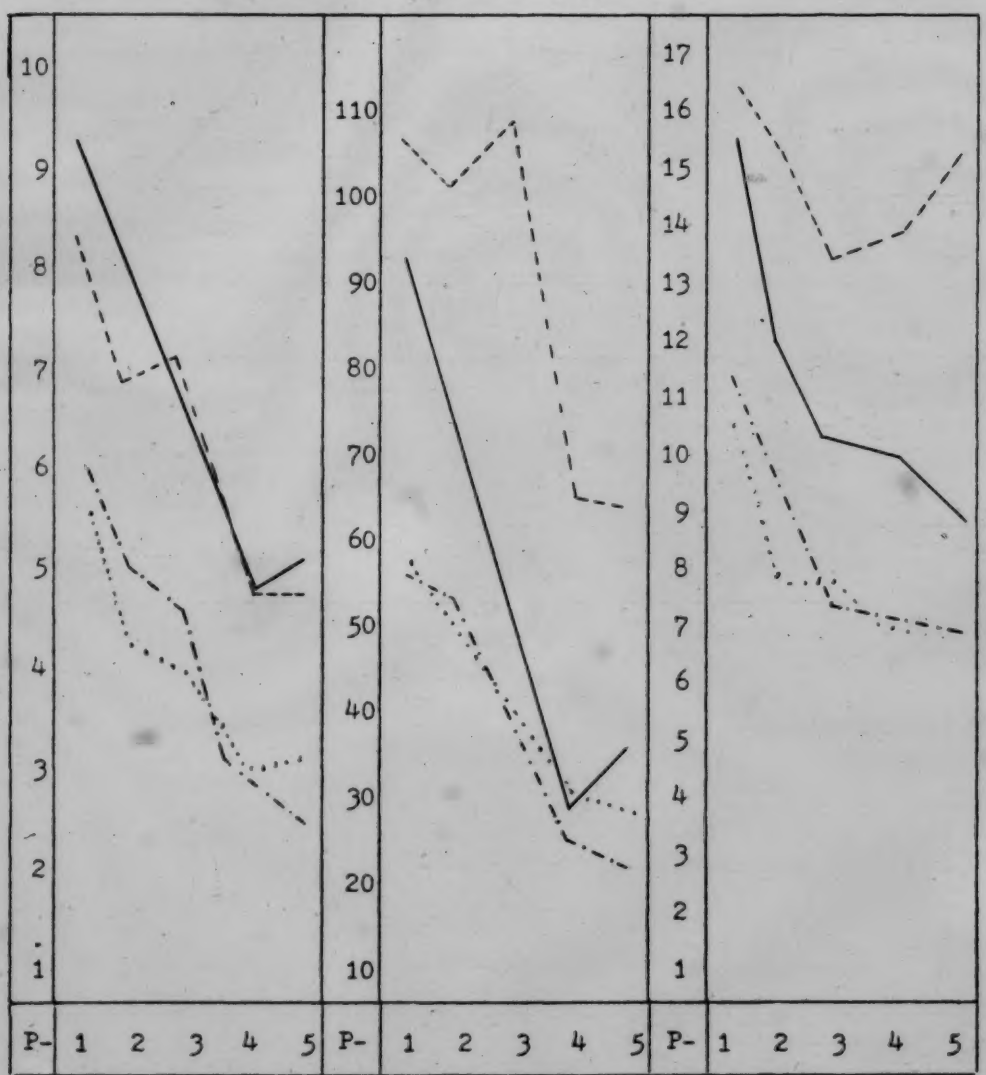


Table 4-2

Headache Activity Variables

Total Groups'--Cell Means

Across Five Repeated Measures

	P-1	P-2	P-3	P-4	P-
Headache Days					
All subjects N=48	7.20	6.00	5.51	3.83	3.72
Headache Hours					
All Subjects N=48	78.49	69.94	61.88	37.81	36.94
Headache Index					
All Subjects N=48	13.35	11.15	9.69	9.39	9.34

Post-hoc Multiple Comparisons (Newman-Keuls)
--lines denote significant differences $p < .01$

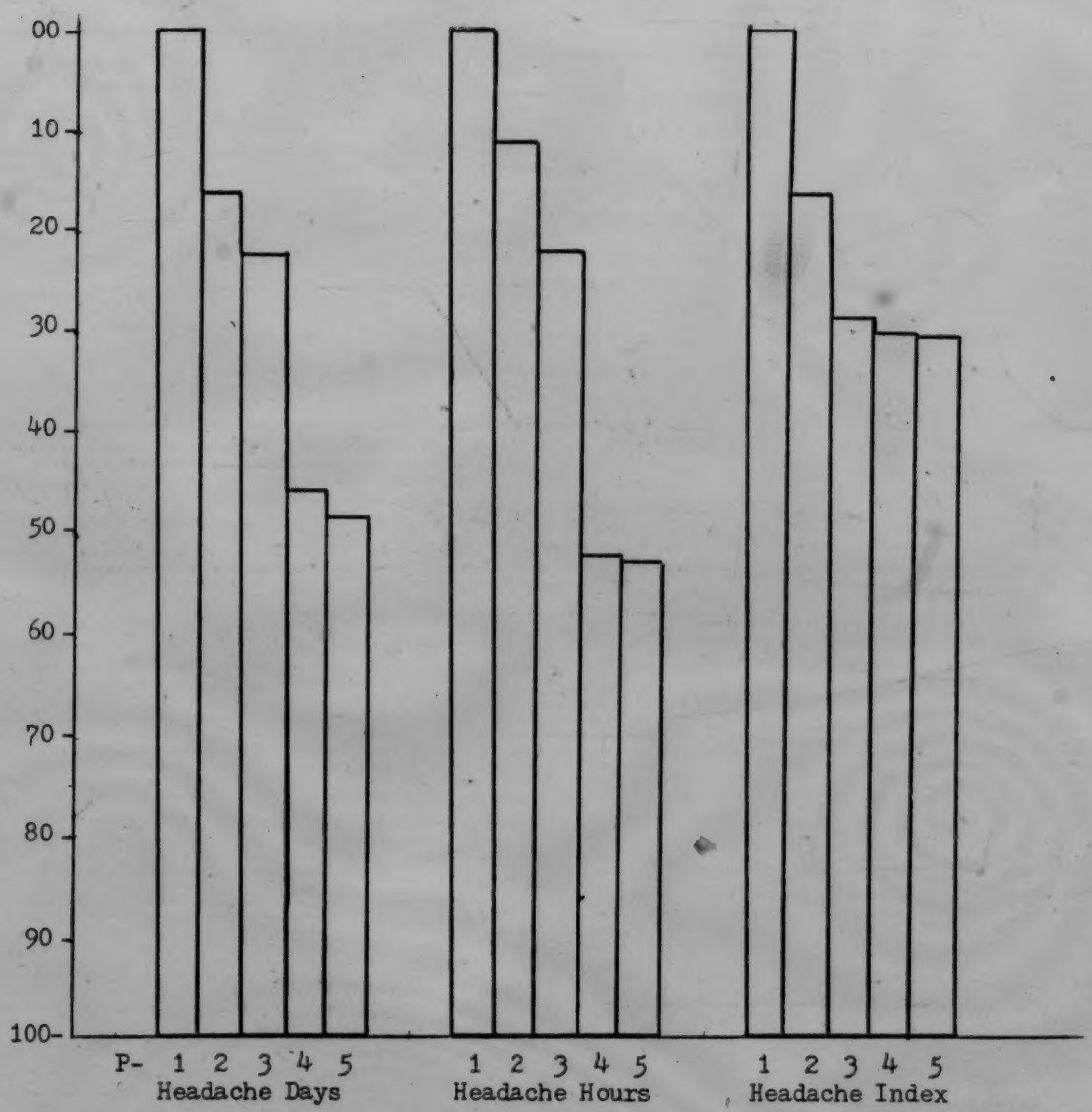
Figure 4-3

Total Groups'--Cell Means

Across Five Repeated Measures

Percentage Improvement

(Headache Activity Variables)



Measures Calculated Over Two, Fourteen Day Periods N = 48

groups were collapsed and the means from P-1 through P-5 were compared to one another, significant differences between the repeated measures emerged on all of the headache activity variables. Concerning the reduction of headache days, differences were noted between pre-treatment and each of the three post-treatment measures, between treatment and each of the three post-treatment measures, and between post-treatment one (P-3) and post-treatment three (P-5). Concerning the reduction of headache hours, differences were noted between pre-treatment and the second and third post-treatment measures, between treatment and the second and third post-treatment measures, and between the first post-treatment measure and the second and third post-treatment measures. Concerning the reduction of the headache index, differences were noted between pre-treatment and each of the three post-treatment measures.

The cell means for the total subject population (N = 48) are presented in Table 4-2 (headache days, headache hours, headache index) and are presented graphically in Figure 4-3.

Recovery Responsivity Groups Analyses of Variance

The headache activity variables were analysed using three two-factor Analyses of Variance with two levels of Factor A and five repeated measures on Factor B. The groups in Factor A were:

slow EMG recoverers, relative to ST recovery (n = 24)

slow ST recoverers, relative to EMG recovery (n = 24)

Table 4-3

Headache Activity Variables

Two Responsivity Groups'--Cell Means

Across Five Repeated Measures

		P-1	P-2	P-3	P-4	P-5
Headache Days						
EMG recovery responsive*	n=24	6.625	5.333	5.188	3.646	3.667
ST recovery responsive**	n=24	7.417	6.333	5.500	3.813	3.542
Headache Hours						
EMG recovery responsive	n=24	78.292	72.542	69.563	44.979	43.604
ST recovery responsive	n=24	73.375	62.708	48.458	27.604	26.750
Headache Index						
EMG recovery responsive	n=24	12.875	10.833	10.021	9.708	10.188
ST recovery responsive	n=24	13.167	10.750	8.771	8.375	7.708

*slow EMG recovery following stressor, during pre-treatment assessment

**slow ST recovery following stressor, during pre-treatment assessment

Figure 4-4

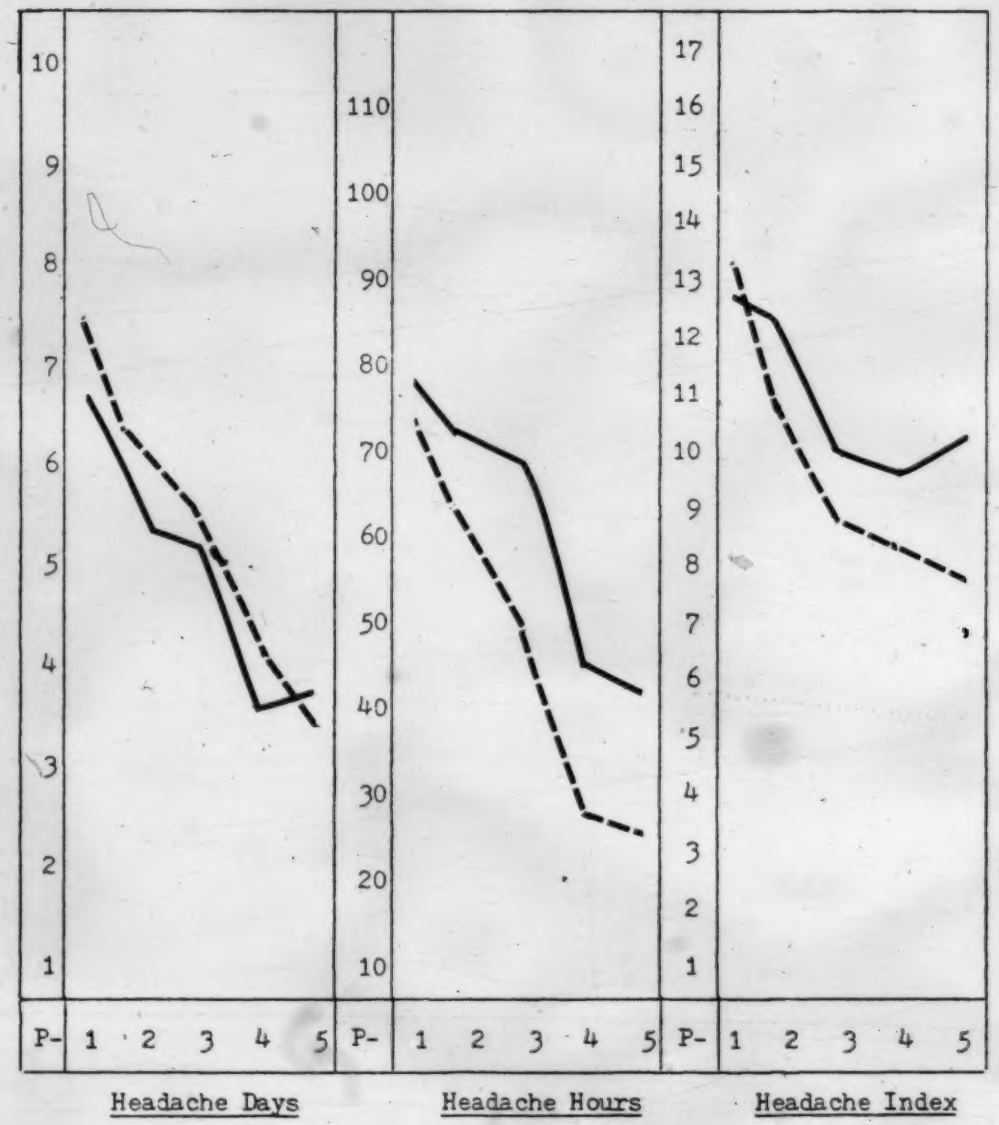
Headache Activity Variables

Two Responsivity Groups'--Cell Means

Across Five Repeated Measures

EMG recovery responsive ————————

ST recovery responsive ----------



A significant period effect was noted when the two groups were collapsed. There were significant reductions in the number of headache days, $F(4,184) = 100.84$, $p < .001$; in the number of headache hours, $F(4,184) = 16227.01$, $p < .001$; and in the summed daily intensity index, $F(4,184) = 142.26$, $p < .001$. The cell means for each recovery responsivity group across five measures are presented in Table 4-3 (headache days, headache hours, headache index) and presented graphically in Figure 4-4. The corresponding Analyses of Variance source tables are in Appendix N. No significant differences were noted between the two groups.

Biofeedback Treatment Groups Analyses of Variance

The headache activity variables were analysed using three two-factor Analyses of Variance with two levels of Factor A and five repeated measures on Factor B. The groups in Factor A were:

EMG biofeedback participants (n = 25)

ST biofeedback participants (n = 23)

A significant period effect was noted when the two groups were collapsed but no between-group differences were significant. There were significant reductions in the three headache activity variables as previously reported, across the five repeated measures. The cell means for each group across five measures are presented in Table 4-4 (headache days, headache hours, headache indes) and presented graphically in Figure 4-5. The corresponding Analyses

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Table 4-4

Headache Activity Variables

Two Treatment Groups'--Cell Means

Across Five Repeated Measures

	P-1	P-2	P-3	P-4	P-5
Headache Days					
EMG Treatment n=25	7.120	5.880	5.160	3.700	3.760
ST Treatment n=23	6.913	5.783	5.543	3.761	3.435
Headache Hours					
EMG Treatment n=25	72.880	60.960	48.220	30.540	31.340
ST Treatment n=23	79.043	74.870	70.739	42.543	39.348
Headache Index					
EMG Treatment n=25	12.640	9.600	8.900	8.180	7.580
ST Treatment n=23	13.435	12.087	9.935	9.978	10.435

Figure 4-5

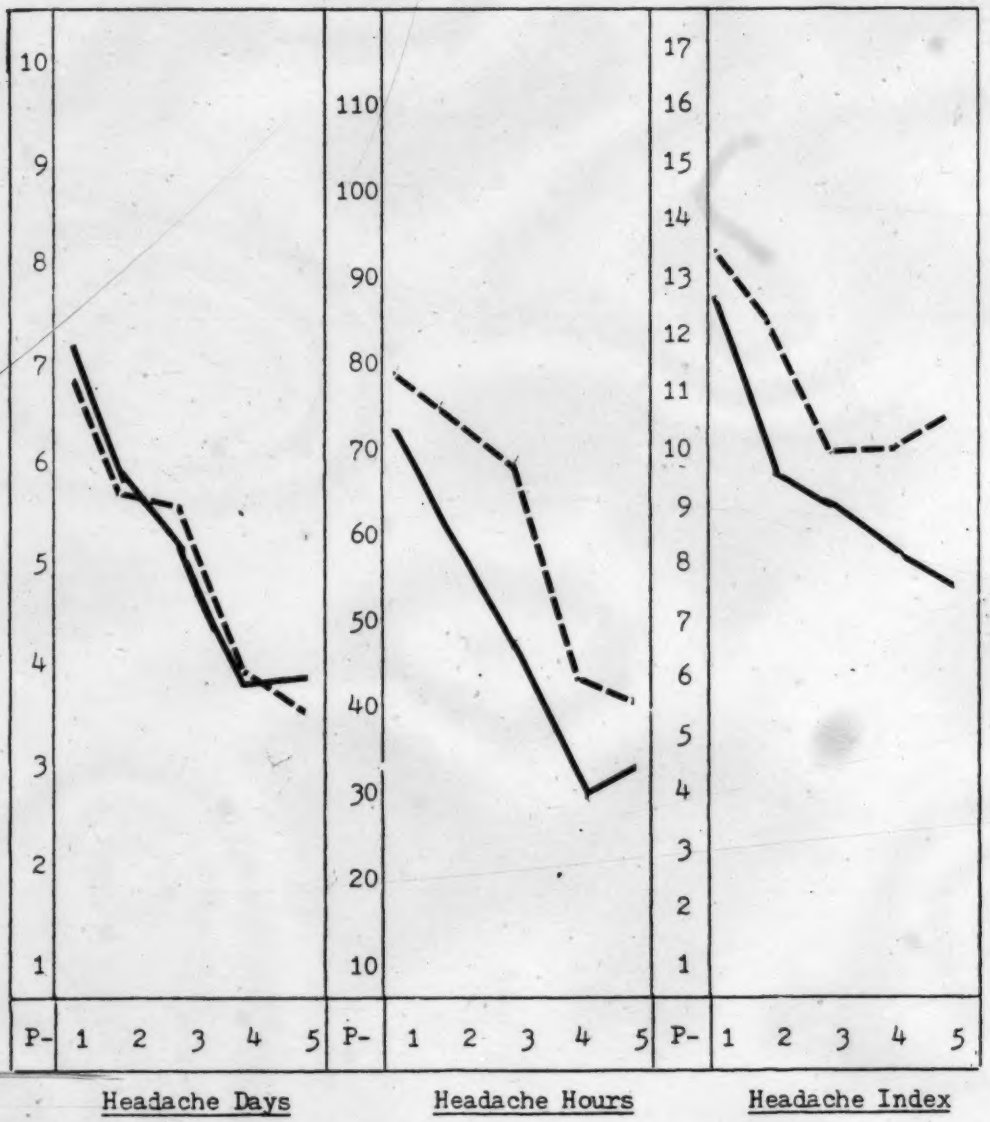
Headache Activity Variables

Two Treatment Groups'--Cell Means

Across Five Repeated Measures

EMG treatment ————————

ST treatment ----------



of Variance source tables are in Appendix N. No significant differences were noted between the two groups.

Matched and Non-Matched Biofeedback Groups Analyses of Variance

The headache activity variables were analysed using three two-factor Analyses of Variance with two levels of Factor A and five repeated measures on Factor B. The groups in Factor A were:

slow EMG recoverers/EMG training, and
slow ST recoverers/ST training (n = 27)

slow EMG recoverers/ST training, and
slow ST recoverers/EMG training (n = 21)

A significant between-groups main effect was noted concerning the reduction of headache days, $F(1,46) = 5.41$, $p < .05$, and concerning the reduction of headache index, $F(1,46) = 4.14$, $p < .05$. The groups did not differ in terms of headache hour reduction. The two groups differed in terms of headache activity variables at the pre-treatment baseline (P-1), but the A X B interaction was not significant indicating that although there were differences at P-1, P-2, P-3, P-4, and P-5, the percentage change over time was not significantly different between the groups.

A significant period effect was noted when the two groups were collapsed. There were significant reductions in the three headache activity variables, as previously noted, across the five repeated measures. The cell means for each group across the five measures are presented in Table 4-5 (headache days, headache hours, headache index) and presented graphically in Figure 4-6. The

Table 4-5

Headache Activity Variables

Matched and Non-Matched Biofeedback Groups'--Cell Means

Across Five Repeated Measures

		P-1	P-2	P-3	P-4	P-5
Headache Days						
EMG/EMG and ST /ST *	n=27	5.704	4.630	4.222	3.000	2.741
EMG/ST and ST /EMG**	n=21	8.714	7.381	6.786	4.667	4.714
Headache Hours						
EMG/EMG and ST /ST	n=27	58.481	53.963	43.278	28.222	24.722
EMG/ST and ST /EMG	n=21	98.143	85.190	79.238	46.667	48.619
Headache Index						
EMG/EMG and ST /ST	n=27	10.815	8.667	7.556	6.944	6.722
EMG/ST and ST /EMG	n=21	15.857	13.524	11.762	11.738	11.810

*EMG recoverers/EMG training
ST recoverers/ST training

**EMG recoverers/ST training
ST recoverers/EMG training

Figure 4-6

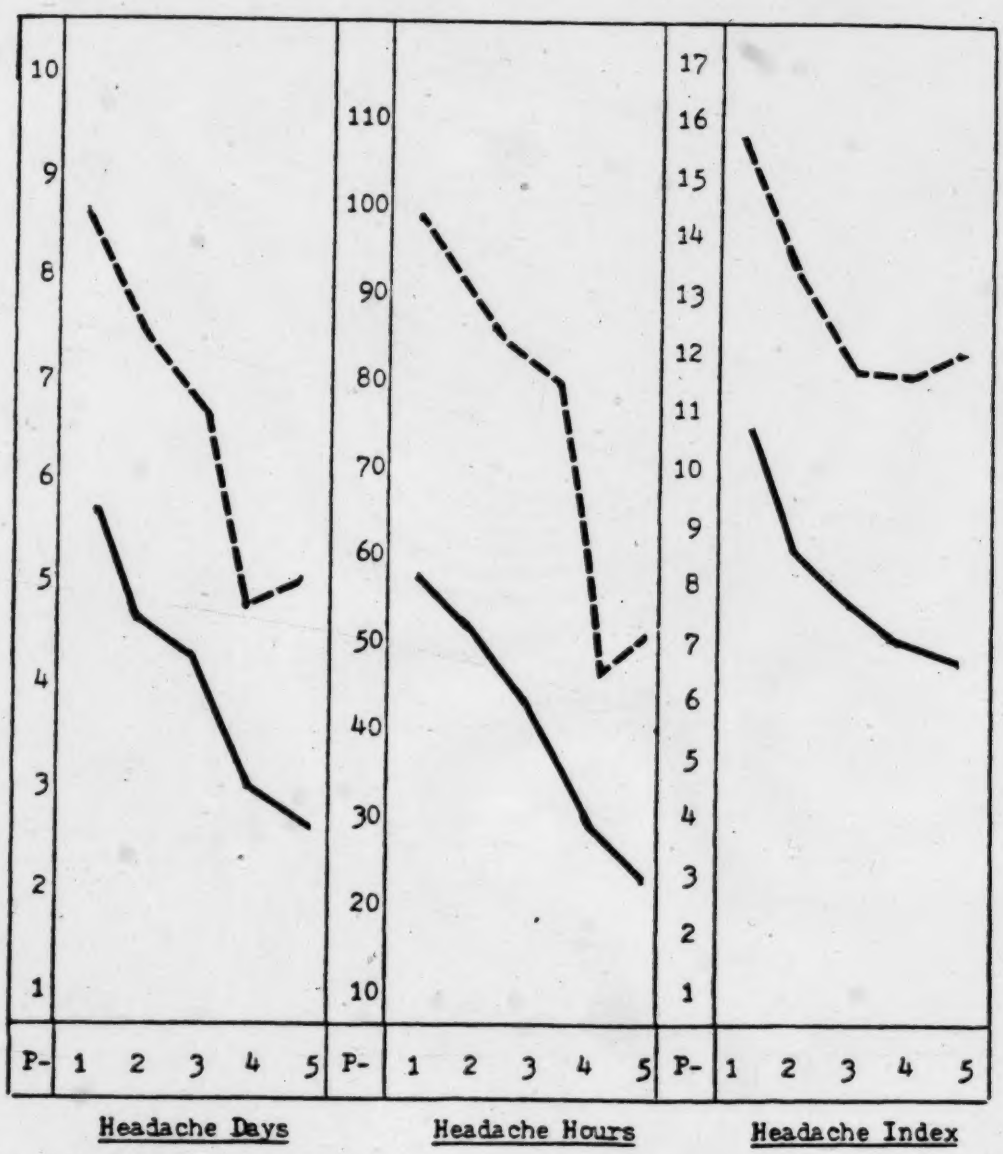
Headache Activity Variables

Matched and Non-Matched Biofeedback Groups'--Cell Means

Across Five Repeated Measures

Matched ————————

Not-Matched ----------



corresponding Analyses of Variance source tables are in Appendix N.

Met Criterion and Not-Met Criterion Groups Analyses of Variance

The headache activity variables were analysed using three two-factor Analyses of Variance with two levels of Factor A and five repeated measures on Factor B. The groups in Factor A were:

Participants who attained specific skill with instrumentation but were not necessarily weaned (n = 32)

Participants who were not able to attain specific skill even with the instrumentation (n = 16)

A significant period effect was noted when the two groups were collapsed but no between-group main effects were indicated. There were significant reductions in the three headache activity variables, as noted, and the cell means for each group across the five repeated measures are presented in Table 4-6 (headache days, headache hours, headache index) and presented graphically in Figure 4-7. The corresponding Analyses of Variance source tables are in Appendix N.

Weaned and Not-Weaned Groups Analyses of Variance

A final series of three two-factor Analyses of Variance was completed with the same headache activity variables across five repeated measures (Factor B). The groups in Factor A were:

Participants who demonstrated specific self-control skill with and without the use of instrumentation (n = 20)

Participants who were unable to demonstrate acquired skill without the use of instrumentation (n = 28)

Table 4-6

Headache Activity Variables

Met Criterion and Not-Met Criterion Groups'--Cell Means

Across Five Repeated Measures

		P-1	P-2	P-3	P-4	P-5
Headache Days						
Criterion Met	n=32	7.813	6.031	5.922	3.781	3.719
Crit. Not Met	n=16	5.438	5.438	4.188	3.625	3.375
Headache Hours						
Criterion Met	n=32	87.531	69.813	65.047	39.141	38.953
Crit. Not Met	n=16	52.438	63.250	46.938	30.594	27.625
Headache Index						
Criterion Met	n=32	14.469	10.813	10.234	8.594	9.234
Crit. Not Met	n=16	10.125	10.750	7.719	9.938	8.375

Figure 4-7

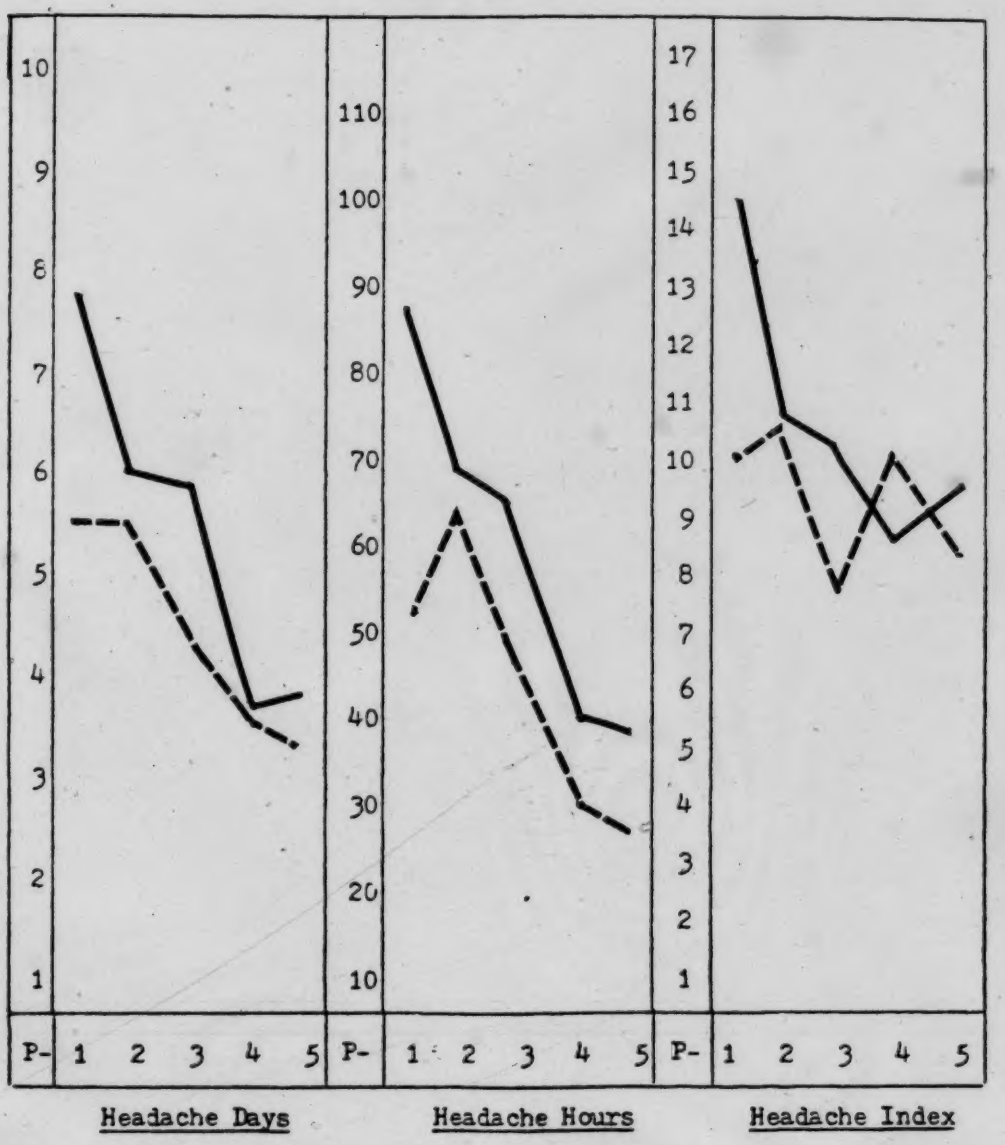
Headache Activity Variables

Met Criterion and Not-Met Criterion Groups'--Cell Means

Across Five Repeated Measures

Criterion Met ————————

Crit. Not Met ----------



The analyses indicated no significant main effect between groups concerning any of the three variables. There were significant period effects (Factor B) in terms of headache days, headache hours, and headache index as previously documented. There were also significant A X B interactions in terms of headache days, $F(4,184) = 5.17$, $p < .001$; headache hours, $F(4,184) = 4.36$, $p < .01$; and headache index, $F(4,184) = 5.44$, $p < .001$. The interactions were such that the group of subjects who had been weaned (proven skill acquisition with and without the use of instrumentation) were not only experiencing greater headache activity improvement during treatment and during the first month of follow-up compared to those who had not been weaned (may have been able to demonstrate skill acquisition with instrumentation but not without the use of instrumentation), but continued to experience marked headache reduction while the not-weaned group showed minimal progress or, in the case of headache index, a return to pre-treatment level. The cell means for the two groups across five repeated measures are presented in Table 4-7 and presented graphically in Figure 4-8. The corresponding Analyses of Variance source tables are in Appendix N. The group X period interaction is presented numerically in Table 4-7.

Post-hoc multiple comparisons using the studentized range, Newman-Keuls method were used to determine where significant differences occurred among the five repeated measures of both the weaned group and the not-weaned group. Table 4-8 indicates the significant differences between pairs of repeated measures; the lines

Table 4-7

Headache Activity Variables

Weaned and Not-Weaned Groups'--Cell Means

Across Five Repeated Measures

		P-1	P-2	P-3	P-4	P-5
Headache Days						
Weaned	n=20	7.500	5.250	5.000	2.425	2.100
Not Weaned	n=28	6.679	6.250	5.589	4.661	4.679
Headache Hours						
Weaned	n=20	84.050	53.950	47.575	20.825	15.150
Not Weaned	n=28	69.966	77.393	67.179	47.339	49.482
Headache Index						
Weaned	n=20	13.550	9.200	8.225	5.725	3.975
Not Weaned	n=28	12.643	11.929	10.232	11.411	12.500

Figure 4-8

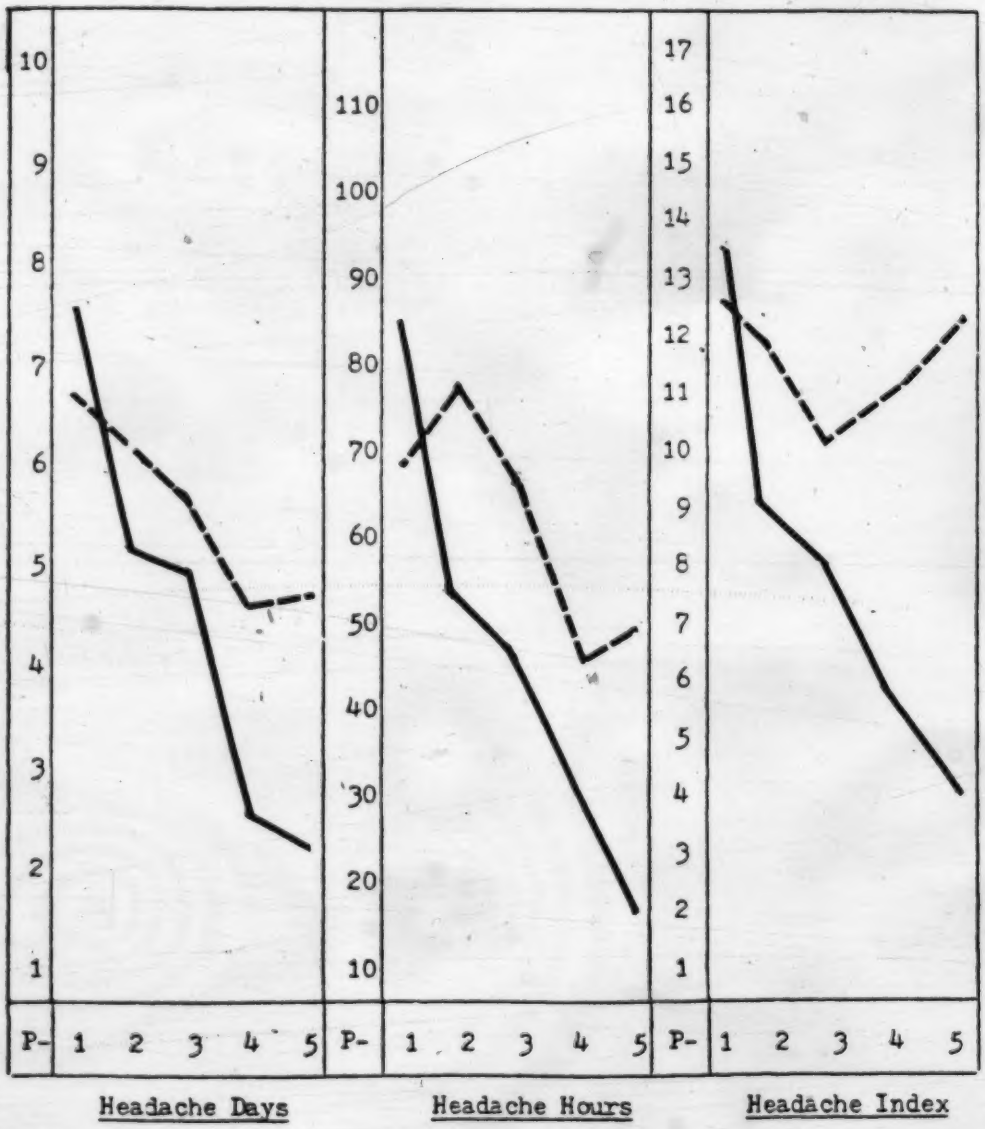
Headache Activity Variables

Weaned and Not-Weaned Groups'--Cell Means

Across Five Repeated Measures

Weaned: ————

Not Weaned ----------



extend from one pair-member to the other indicating that those two measures are significantly different at $p < .01$. The weaned group (n=20) made greater headache activity improvement than the not-weaned group at the point of treatment and at each of the three post-treatment measures. There were significant differences between period measurements in terms of headache days, headache hours, and headache index to the extent that the weaned group continued to demonstrate significant improvement even after the first post-treatment measurement while the not-weaned group experienced continued improvement to a limited extent. Refering to Table 4-8, it is noted that there are more significant pair-wise comparisons within the weaned group than within the not-weaned group and that the headache index variable indicates a remarkable difference in the trends of the two groups.

The weaned and not-weaned groups were evaluated to determine the extent to which each experimental treatment group comprised these two groups. Table 4-9 provides this information. The difference between the number of EMG trained and ST trained subjects should be noted (14 and 6, respectively). The small number of EMG responsive/ST trained and ST responsive/ST trained subjects (3 + 3 = 6), also needs to be noted. Further analyses with as few subjects within cells would not have provided meaningful information.

Table 4-8

Weaned and Not-Weaned Groups' Changes Over Time: Represented as a Percentage of Baseline

		P-1	P-2	P-3	P-4	P-5
Headache Days						
Weaned	n=20	100.0	70.0	66.7	32.3	28.0
Not Weaned	n=28	100.0	93.6	83.7	69.8	70.1
Headache Hours						
Weaned	n=20	100.0	64.2	56.6	24.8	18.0
Not Weaned	n=28	100.0	110.6	96.0	67.7	70.7
Headache Index						
Weaned	n=20	100.0	67.9	60.7	42.2	29.3
Not Weaned	n=28	100.0	94.4	80.9	90.2	98.9

Post-hoc Multiple Comparisons (Newman-Keuls)
-lines denote significant differences, $p < .01$

Table 4-9

Weaned and Not-Weaned Groups:

Member Breakdown

			Experimental Group	
Weaned n = 20				
Recovery Responsive				
EMG	(1)	n = 11	1/1	n = 8
ST	(2)	n = 9	1/2	n = 3
Treatment Received			2/1	n = 6
EMG	(1)	n = 14	2/2	n = 3
ST	(2)	n = 6		20
Not Weaned n = 28				
Recovery Responsive				
EMG	(1)	n = 13	1/1	n = 6
ST	(2)	n = 15	1/2	n = 7
Treatment Received			2/1	n = 5
EMG	(1)	n = 11	2/2	n = 10
ST	(2)	n = 17		28

Profile Analyses

Multivariate Profile Analyses were used to assess group trends during the post-treatment period (P-3 to P-5). Group means were plotted and statistical tests determined whether the resulting group profiles differed significantly during the post-treatment period.

Although subjects had been monitored for forty-eight weeks after treatment, only the first forty weeks were analysed due to program limitations (Division of Educational Research Services, Faculty of Education: Multivariate Profile Analysis, Mulv14).

A series of profile analyses were computed using a number of statistical tests but as in the Analyses of Variance for repeated measures only the weaned/not-weaned factor A grouping resulting in significant differences other than the consistent period main effect.

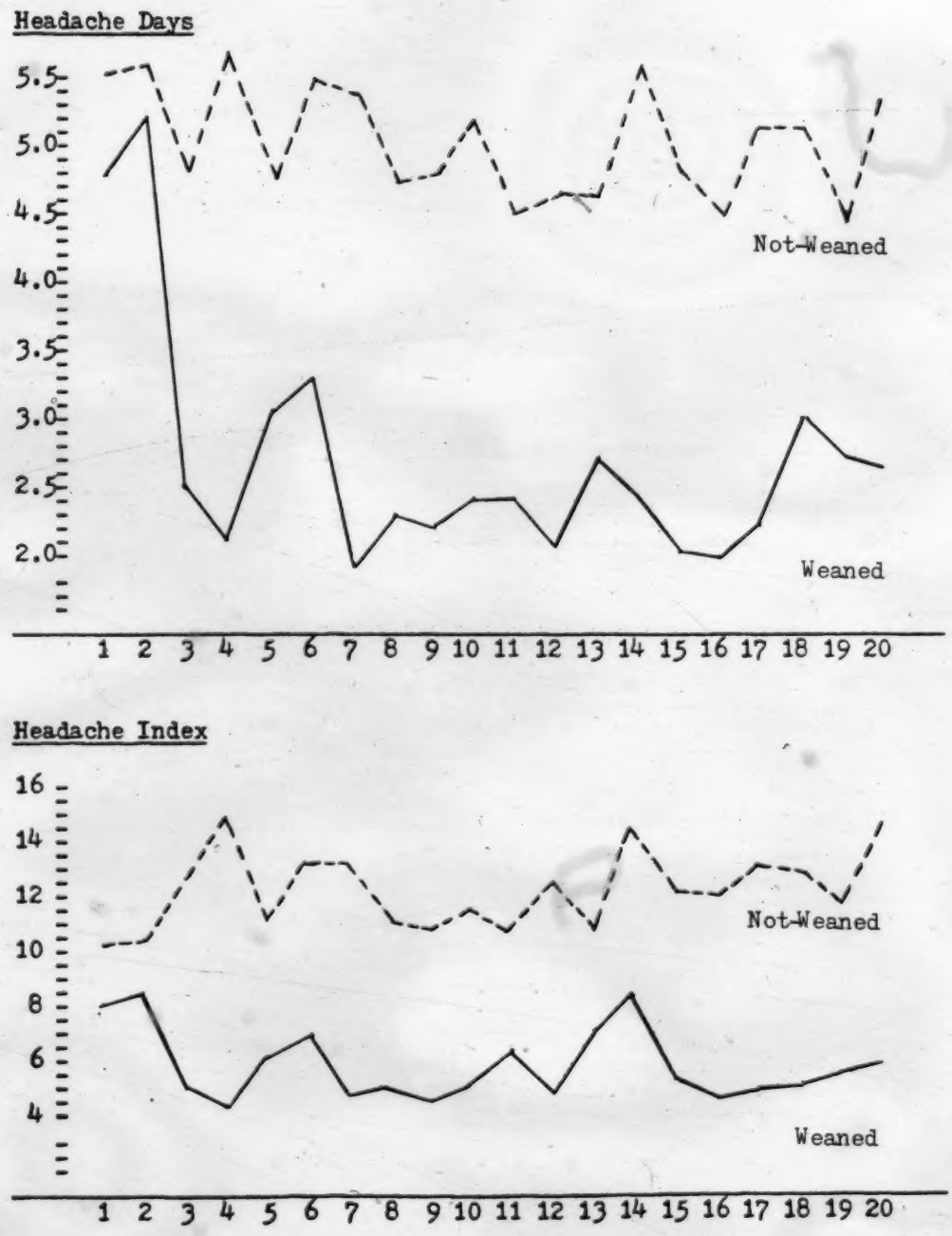
Weaned and Not-Weaned groups differed significantly in terms of headache days and headache index. For both measures there were significant differences between group means calculated over the twenty variables, headache days $F(1,46) = 4.53$, $p < .05$; headache index $F(1,46) = 5.02$, $p < .05$. Figure 4-10 indicates these differences, and primarily provides further substantiation of the Analyses of Variance results. These Profile Analyses provide detailed representations of the trends from P-3 to P-5, as previously discussed.

Figure 4-10

Profile Analyses

Twenty, Fourteen-Day Measures from

Termination of Treatment



Relaxation Exercises

Upon beginning treatment each participant was instructed to practise relaxation techniques daily, and were encouraged to continue such endeavours throughout the follow-up. Analyses of Variance completed with the various Factor A arrangements explained earlier indicated that only the Weaned and Not-Weaned Groups differed significantly in the amount of relaxation done from P-2 to P-5.

When all groups were collapsed there was an apparent decrease in the amount of practise done as the follow-up period continued:

P-1	000.00	minutes per fourteen day period per individual
P-2	246.60	minutes per fourteen day period per individual
P-3	219.59	minutes per fourteen day period per individual
P-4	106.01	minutes per fourteen day period per individual
P-5	91.38	minutes per fourteen day period per individual

The Multivariate Profile Analyses used to assess the group differences during post-treatment (forty weeks following end of treatment), indicated that the weaned and not-weaned groups differed significantly in terms of minutes of relaxation completed $F(1,46) = 5.56$, $p < .05$. The weaned group reportedly did fewer minutes of relaxation from P-2 to P-5, than did the not-weaned group.

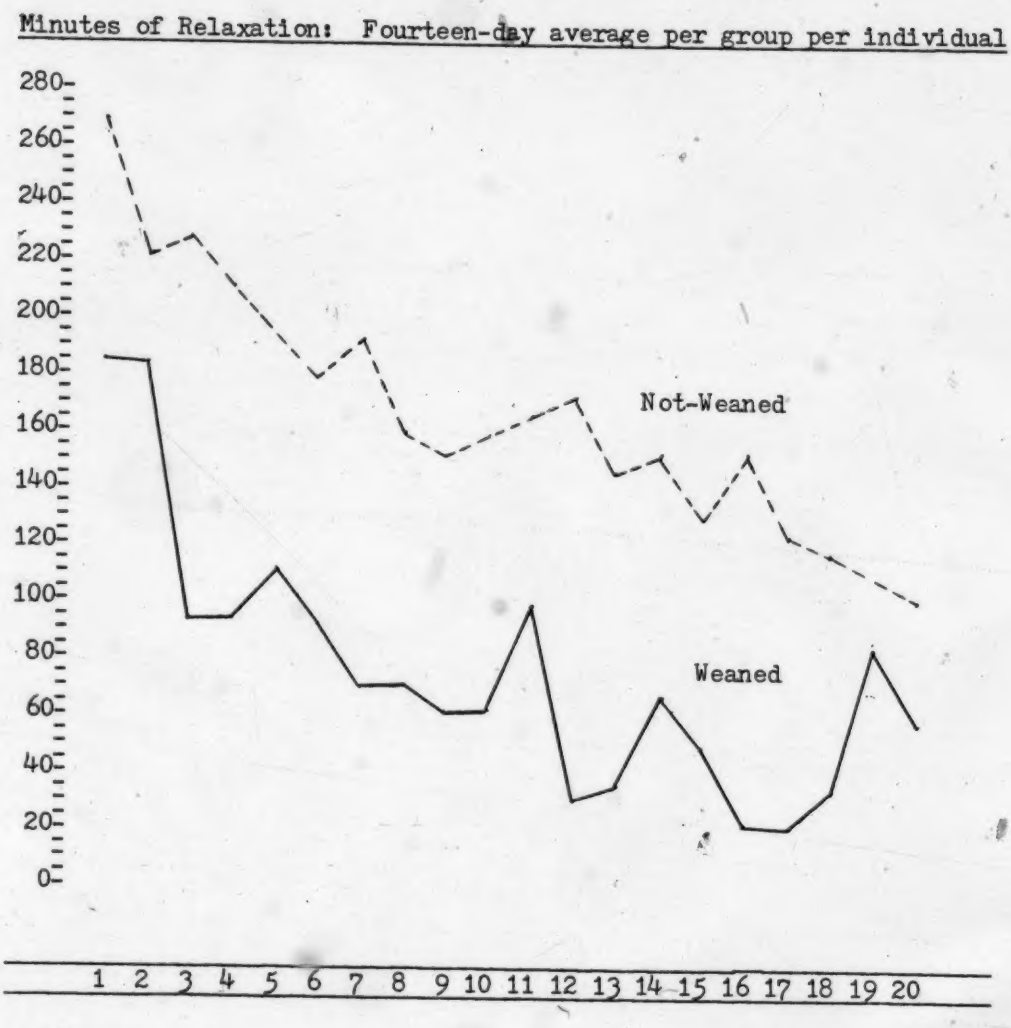
These differences are graphically presented in Figure 4-11 across the forty weeks following end of treatment.

Figure 4-11

Profile Analyses

Twenty, Fourteen-Day Measures from

Termination of Treatment



Descriptive Data

Perhaps the most meaningful information that can be provided by this research in terms of clinical application, would be a presentation of how many subjects from each grouping actually experienced what might be considered "considerable improvement". Convention suggests that "considerable improvement" is that reduction in headache activity that is at least fifty percent from the baseline information. Table 4-10 provides this information. The data presented is the Headache Index and is a measure of the percentage improvement from the pre-treatment baseline to the last four weeks of the long-term follow-up, calculated as follows:

$$\frac{\text{Average headache index for four weeks of pre-treatment baseline} - \text{Average headache index for last four weeks of follow-up}}{\text{Average headache index for four weeks of pre-treatment baseline}}$$

The calculation was completed by multiplying the arrived at number by 100, to provide the percentage improvement from pre-treatment to end of long-term follow-up. The difference between the Weaned and the Not-Weaned Groups have already been presented as being significant. The differences become more meaningful when the breakdown is done as in Table 4-10.

It needs to be explained that since the comparison-wise F-tests that were significant were all significant at the .01 alpha-level and occasionally at the .001 alpha-level. This means that although six Anovas were completed, the overall experiment-wise alpha-level was still $p < .05$.

Table 4-10

Percentage Improvement of

Headache Index

Pre-Treatment to End of Follow-Up

Group	n	Unimproved or Worse (<20%)	Somewhat (20%-49%)	Much Improved (+50%)
EMG/EMG	14	35.7%	14.3%	50.0%
EMG/ST	10	40.0%	20.0%	40.0%
ST/EMG	11	18.2%	18.2%	63.6%
ST/ST	13	30.7%	7.7%	61.6%
Weaned	20	10.0%	15.0%	75.0%
Not-Weaned	28	46.4%	14.3%	39.3%

CHAPTER FIVE

DISCUSSION

Three questions were stated at the close of Chapter Two. Each will be addressed in turn, having been statistically described in the previous chapter. The implications of each will be discussed with particular attention given to suggestions for long-term therapeutic benefits in the treatment of migraine headaches.

Research Question One

1. Is there a difference between training a migraineur on the modes that have been found to be slow or quick to recover following the removal of a psychosocial stressor?

Four separate series of analyses of variance were computed to answer this question. The first series (Experimental Treatment Groups) indicated that although all four treatment groups (EMG recovery responsive/EMG treatment; EMG recovery responsive/ ST treatment; ST recovery responsive/EMG treatment; ST recovery responsive/ST treatment) had significant decreases in the number of

headache days, in the number of headache hours, and in the headache index throughout the year, there were no differences between the four groups in terms of these three headache activity variables. One should note that the EMG/ST group's (n = 10) headache index increased following the one month follow-up such that at the end of the research, that measure had returned to the level experienced during treatment. When the four groups were collapsed and headache activity variables were assessed from the total group (N = 48), it is evident that in terms of headache days (per fourteen day period), significant improvement occurred not only from the pretreatment to treatment and post-treatment, but also from the beginning of posttreatment follow-up to the end of posttreatment follow-up. The same continuation of improvement was evident in terms of headache hours but did not occur in terms of headache index. In terms of the headache index, subjects did not improve beyond what they had gained by the one-month follow-up. This is, however, a total group measure and was influenced by the poor showing of the EMG/ST group.

Again, there were no between-group differences noted but all groups did manifest a reduction in headache activity although not all groups appeared to uphold this improvement throughout the follow-up.

The second series of analyses of variance (Recovery Responsivity Groups) indicated that although continued improvement was evident throughout the year on all three headache activity variables, the two groups did not differ.

The third series of analyses of variance (Treatment Groups) similarly indicated that although continued improvement was evident throughout the year on the three headache activity variables, the two groups did not differ. Thus far it appears that the subjects did not differ among themselves regardless of the groups being investigated, that EMG treatment and ST treatment were essentially equally effective and were as effective regardless of the recovery responsive group receiving the particular treatment.

The fourth series of analyses of variance (Matched vs Non-Matched Groups) supported the mentioned improvement over time and indicated a between groups difference in terms of headache days and the headache index. However, these differences were constant from the time of pretreatment baseline through to the end of the long-term follow-up, indicating that the difference between the two groups did not change (no interaction, statistically) and that although randomization procedures were followed, the matched group (n = 27) differed from the non-matched group (n = 21) before treatment was initiated.

The conclusion is that with this particular population of migraine headache sufferers the two treatment approaches did not have differing effectiveness even when given to subjects who had been predetermined as likely to benefit more from one treatment than the other. The exciting result is that dramatic improvements were evident across the year for all subjects in terms of headache days per fourteen day period, in terms of headache hours per fourteen day period, and in terms of the headache index for three of the

four experimental groups. The fourth group was determined as being slow EMG recovery responsive following the stressor during the physiological profile, and had received ST treatment. Not only was the improvement as a result of treatment minimal compared to the other groups but, after the one-month follow-up, headache activity increased (worsened) as assessed by the headache index. The difference however, was not statistically significant and further research would be necessary to assess the consistency of such a trend.

It must be noted that while the subjects recorded hourly headache information up until the end of the first follow-up month, data gathered subsequently was done so on a daily basis. There is no information presently available indicating whether hourly and daily gathering of such a self-report measure as head pain, is comparable. Until such is available, further research may be advised to use daily ratings of subjective discomfort consistently during research.

There have been some limitations to the present study that need to be realized at this point, that might cast further light onto the present findings. First, subjects were divided into two groups (slow EMG recovery responsive compared to ST measure, slow ST recovery responsive compared to EMG measure, following the removal of psychosocial stressor during profile assessment session). This division was done such that all subjects were included in one of the groups, divided by way of a median split. This means that some subjects who may have been almost identical in terms of these physiological measures found themselves in different groups. Further research may rather choose to omit the middle, similar subjects, and

make use of the more dissimilar subjects. In so doing, the two groups would be less similar and would more likely demonstrate their differential characteristics without the averaging-out effects of the subjects who clustered around the mean but ended up being assigned to different groups. Second, there is recent evidence from the University of Alberta (Meen, 1981) suggesting that migraineurs and non-migraineurs may not differ on the two physiological measures utilized in this study. Meen reported that in terms of EMG and ST characteristics following a psychosocial stressor, the migraineur did not differ from the non-migraineur and it follows that preference for one type of biofeedback training over another may not be warranted. Further research is needed to assess possible differences between migraineurs and non-migraineurs, between migraineurs and muscle contraction headache sufferers, and among migraineurs themselves.

Research Question Two

2. Is there a benefit to training a migraineur to criterion that is reflected in a reduction in headache activty (with instrumentation available)?

The analyses of variance indicated no significant differences were found between the two groups. Individuals who had been able to reach a specific level of control with the aid of biofeedback instrumentation did not differ from those who had not been able

to demonstrate specific control with the instrumentation. This suggests that monitoring one's headache activity, learning some relaxation techniques, and practising those strategies at home may be more important than being able to demonstrate control with biofeedback instrumentation present--that being able to control one's physiology in a direct and observable way may not be as important and essential as is learning some basic relaxation techniques.

Research Question Three

3. Is there a benefit to training a migraineur to the point where competency can be demonstrated with and without the aid of instrumentation, that is reflected in a reducation in headache activity?

The analyses of variance computed with the two groups of Factor A being Weaned and Not-Weaned ($n = 20$; $n = 28$, respectively), indicated that although there were no main effects between the groups (means did not differ significantly), there was the above mentioned among-groups period effect. There was an over-all improvement in the total groups, and most importantly, there were significant interactions on all three headache activity variables. The group of subjects who had been able to demonstrate self-regulation without the instrumentation being available (the weaned group), was not only experiencing fewer headaches at the time of treatment and during the first four weeks of follow-up after the termination of treatment,

but continued to show marked improvement in headache reduction compared to the group of subjects who had not been able to demonstrate self-regulation without the use of instrumentation. Although headache days and headache hours improved over the year for both groups, the weaned group's improvement was significantly greater. In terms of the headache index, the conventional measure of headache activity, the weaned group continued to improve markedly while the not-weaned group's rating of the headache index returned to pretreatment baseline levels.

This is a most exciting difference particularly in view of the profile analyses of the first forty weeks following termination of treatment. In terms of headache days the not-weaned group appears to have made no changes from the first week of follow-up to the fortieth week, compared to the weaned group which dropped markedly, and essentially held that drop in days of headache throughout the forty weeks. The two groups experienced 7.50 (weaned) and 6.68 (not weaned) headache days per fourteen day period before treatment and 5.00 (weaned) and 5.59 (not weaned) headache days per fourteen day period during the first month after termination of treatment. Forty weeks later the weaned group had an average approximately half that of the not-weaned group (2.5 headache days compared to 5.25 headache days).

In terms of the headache index, the differences are not as marked but are never-the-less as significant. These differences, supported by both the analyses of variance and the profile analyses are most interesting in light of the differences between the two

groups in the amount of relaxation exercises done throughout the follow-up period. The not-weaned group did more at-home relaxation practise than did the weaned group. One might assume that the not-weaned group experienced greater headache activity (lesser degree of improvement) and so maintained their at-home practise in an effort to further reduce the headaches, and yet, were still not able to bring about the changes as experienced by the weaned group.

This suggests that while emphasis has usually been placed on whether a patient is receiving the most appropriate training depending on the nature of the problem, and on the amount of time the individual is able to devote to the clinic training and at-home practise, and perhaps on instilling in the patient a sense of mastery, of self-efficacy, the more appropriate focus would be to determine whether learning has actually occurred. It is perhaps incorrect to assume that any therapeutic benefit will result unless skill acquisition has been clearly established. The number of sessions is clearly not an important factor in itself because, in the present study, the weaned group of subjects had fewer clinic visits, (group average = 8 sessions), than did the not-weaned group (group average = 12 sessions).

Unless concrete evidence is available to prove that learning has taken place, one ought not to assume that it has been acquired. Townsend (1975) reported that the EMG readings obtained fourteen days after end of treatment (frontalis, for chronic anxiety), were not significantly lower than those of the control group (group therapy, for chronic anxiety). Transfer of the skill beyond the

training sessions had not occurred. Weiss (1975) reviewed Townsend's work and suggested that a weaning procedure would facilitate learning and maximize the likelihood of transfer of the skill beyond the clinical setting. Weiss and Engel (1971) had employed a weaning procedure in the biofeedback training of cardiovascular patients with premature ventricular contractions and reported improved cardiovascular function for longer periods of time, as a result. The present study provides further evidence that unless a skill has been proven, the ability may not be firmly established within the client's repertoire.

The implication for the practising clinician is that when approached by a distressed individual, the promise of biofeedback should be qualified with the statement that individuals are unique, with different capacities for acquiring skills and with different rates of learning those skills. Rather than suggest that ten or twelve sessions should prove to be enough training to ensure relief from discomfort, the clinician would be more honest if biofeedback training was presented as a way of learning how to self-regulate one's physiological states and that this learning might take as few as six or eight sessions or as many as twenty or more sessions. When biofeedback training has not been helpful after ten or twelve sessions, clients have left treatment discouraged and clinicians have questioned the original diagnosis, the utility of existing theories of biofeedback mechanisms, and the motivation of the client. The correct stance at that point might be rather to discuss the possibility that prolonged treatment might be necessary,

and whether clinician and client are prepared to carry on with the procedures, realizing that the uniqueness of the client makes it impossible to predetermine how many sessions might be necessary before the skill will be sufficiently well entrenched to be of therapeutic benefit.

This research paper has presented one potentially important and yet perhaps unexplained result, that when trained to a specific level of skill that can be demonstrated even without the aid of biofeedback instrumentation, migraine headache sufferers report much more favourable improvements in their headache activity than their counter-parts who had not been able to demonstrate skill acquisition to the same degree. Weaned subjects had a much higher percentage of 'much improvement' (+50% reduction in headache index) than did the not-weaned group, 75 percent and 40 percent, respectively. The not-weaned group had a much higher percentage of 'unimproved' subjects (<20% reduction in headache index) than did the weaned group, 46 percent and 10 percent, respectively. Moderate improvement (20 to 49 percent reduction in headache index) was 15 percent and 14.3 percent for the weaned and the not-weaned groups, respectively.

Physiological explanations need to be entertained as well as cognitive interpretations of these results. Perhaps the subjects who were able to demonstrate that a specific skill was within their repertoire had in fact learned how to initiate change within their bodies, and had learned how to do it without needing instrumentation. These people may have also had a cognitive advantage over their not-weaned counterparts, for since they were aware that they had acquired

a skill and that instrumentation was not necessary for them to bring about the desired changes, they may have been more likely to attempt to deal with migraine headaches throughout the follow-up, confident that they could deal effectively with them. The individuals who had not proven to themselves and the researchers that they had actually learned a specific skill may have been less confident in their ability to deal effectively with their headaches and may have had difficulty "relaxing in confidence".

It follows then that whether the explanation for the results presented here is primarily a physiological-learning account or a confidence and sense of mastery over physiological states, both are likely to play important roles as the migraineur deals with his or her headaches.

Migraine sufferers are unique. Family histories differ in terms of the number of relatives who may have had migraines. Triggers differ from one individual to another with some being more prone to respond with a migraine headache to foods, some to hormonal fluctuations, some to physical exertion, and some to psychosocial stressors. The actual migraine headache itself may have very similar vasculature characteristics from one migraineur to another but is triggered differently and experienced differently. The biochemical nature of migraine was outlined indepth in Chapter Two as were a number of treatment approaches for migraine headaches. It is only logical that since different treatment approaches (pharmacological, behavioural, cognitive) are effective to varying degrees with different people, even biofeedback training should have considerable variation in its

effectiveness. The clincian needs to be prepared to tailor the treatment for each individual, ready to acknowledge that learning will occur at varying rates for different people. While the exact nature of the biofeedback success with migraines is still debated and the pharmacological approach is often the simplest (when headaches are infrequent and respond readily to medication), it is the responsibility of the clinician to provide a treatment that is effective while at the same time endeavouring to ascertain the physiological components of biofeedback as a treatment for migraine headaches.

Limitations of the Present Study

There are a number of limitations that need to be understood in order that future research in this area might arrive at further answers in the continuing attempt to maximize behavioural treatment and its effectiveness with such dysfunctions as migraine headaches.

1. There is some reason to belief that the forty-eight subjects included in the long-term project were not all pure migraine sufferers. Some may have been suffering from a combination of migraine headaches and muscle contraction headaches. Although these "mixed" headache sufferers were supposedly excluded from the research from the beginning and the researchers relied on a number of criteria (Appendix A), including a migraine diagnosis from a physician, not all subjects were pure migraine headache sufferers. This inadvertent inclusion of some mixed headache sufferers should be noted.

2. The median-split procedure whereby subjects were determined to be slow recoverers on one physiological system relative to another (EMG relative to ST; ST relative to EMG), poses some problems in interpreting the outcome of treatment. In the future it may be more appropriate to conduct a similar treatment project with two groups that are less similar than were the two in this research. Many subjects were not too different from one another and yet were assigned to different groups. A cleaner distinction might be made by assigning to different groups after having dismissed the middle segment of the subject population, those who clustered around the mean-line.

3. The use of hourly headache activity ratings during the first four months of the project and then the use of daily headache activity ratings during the last eight months is perhaps a confounding factor. There is no evidence at present supporting the comparability of these two different types of ratings and there is some question as to the validity of some of the hourly ratings. Where validity was questioned, subjects' data were not included. However, consistent (daily) ratings are recommended throughout the duration of a research project both in the interest of maintaining a consistently acquired measure of headache activity and in the interest of maintaining subject involvement and being able to be confident in the validity of self-report measures. Hourly ratings for four months is a demanding assignment.

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Implications for Further Research

1. Physiological variability among migraineurs needs to be further assessed using different types of stressors, perhaps over a number of physiological stress-profile sessions. Although most migraine sufferers may benefit equally from EMG biofeedback training or ST biofeedback training, there may be the atypical migraineur who would benefit from one treatment modality over another.

2. The reported finding concerning the greater improvement for weaned subjects compared to not-weaned subjects needs further investigation. Setting different levels for non-instrument-control of physiological variables may reveal different degrees of long-term therapeutic benefit, and this needs to be determined. The criteria for skill acquisition was somewhat arbitrarily set in the present study and other more and less difficult criteria should be evaluated.

3. The weaned and not-weaned groups differed in terms of headache activity improvement throughout the follow-up. What needs to be assessed is whether they differed in terms of physiological states and changes as well, particularly those variables that are part of the migraine syndrome (temporal and supraorbital arteries, release of serotonin, uptake of serotonin and excretion in urine, hand temperature, forehead temperature, EMG levels), at time of termination of treatment and at end of long-term follow-up. This would help determine the role that physiological and cognitive factors play in the relief of migraine, particularly the relief as evidenced in the weaned group of subjects.

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BIBLIOGRAPHY

Ad Hoc Committee on Classification of Headache. Classification of headache. American Medical Association Journal, 1962, 179, 717-718.

Adams, H.E. and Feuerstein, M. and Fowler, J.L. Migraine headache: review of parameters, etiology and intervention. Psychological Bulletin, 1980, 87, 217-237.

Adler, C.S. and Adler, S.M. Biofeedback - psychotherapy for the treatment of headaches: A 5-year follow-up. Headache, 1976, 16, 189-191.

Adler, C.S. and Morrissey-Adler, S. Biofeedback and psychosomatic disorders. In J.V. Basmajian (Ed), Biofeedback - Principles and Practice for Clinicians. Baltimore: Williams and Williams Company, 1979.

Alexander, A.B. and Smith D.D. Clinical applications of EMG biofeedback. In R.J. Gatchel and K.P. Price (Eds.), Clinical Applications of Biofeedback: Appraisal and Status. Toronto: Pergamon Press, 1979.

Anthony, M. and Lance, J.W. The role of serotonin in migraine. In Modern Topics in Migraine, J. Pearce (Ed), London: William Heinemann Medical Books Ltd., 1975.

Attfield, M. and Peck, D.F. Temperature self-regulation and relaxation with migraine patients and normals. Behavior Research and Therapy 1979, 17, 591-595.

Bakal, D.A. Headache: A biopsychological perspective. Psychological Bulletin, 1975, 82, 369-382.

Bakal, D.A. Psychology and Medine. New York: Springer Publishing Co., 1979.

Bakal, D.A., Demjen, S. and Kaganov, J.A. Cognitive behavioral treatment of chronic headache. Headache, 20, 163, 1980.

Bakal, D.A. and Kaganov, J.A. Muscle contraction and migraine headache: Psychophysiologic comparison. Headache, 1977, 17, 208-215.

Baldwin, J.S. Effects of thermal feedback training and preheadache are identification on migraine management. Dissertation Abstracts International, 1980, 40, 3916

Bandura, A. Self-efficacy: toward a unifying theory of behavioral change. Psychological Review, 1977, 84, 191-215.

Barabaoz, A.F. and McGeorge, G.M. Biofeedback, medicated biofeedback and hypnosis in peripheral vasodilation training. American Journal of Clinical Hypnosis, 1978, 21, 28-37.

Barlow, D.H. On the relation of clinical research to clinical practice: Current issues, new directions. Journal of Consulting and Clinical Psychology, 1981, 49(2), 147-155.

Basmajian, J.V. Principles and Practice for Clinicians. Baltimore: Williams and Wilkins, 1979.

Bergin, A.E., and Lambert, M.J. The evolution of therapeutic outcomes. In S.L. Garfield and A.E. Bergin (Eds.). Handbook of Psychotherapy and Behavior Change. New York: John Wiley and Sons, 1978.

Bild, R. and Adams, H.E. Modification of migraine headaches by cephalic blood volume pulse and EMG biofeedback. Journal of Consulting and Clinical Psychology, 1980, 48(1), 51-57.

Birbaumer, N. and Kimmel, H.D. Biofeedback and Self-Regulation. New Jersey: Laurence Erlbaum Associates, Publishers, 1979.

Blanchard, E.B. and Ahles, T.A. Behavioral treatment of psychological disorders. Behavior Modification, 1979, 4, 518-549.

Blanchard, E.B., Andrasik, F., Ahles, T.A., Teders, S.J. and O'Keefe, D.D. Migraine and tension headache: a meta-analytic review. Behavior Therapy, 1980, 11, 613-631.

Blanchard, E.B., Andrasik, F., Neff, D.F., Jurish, S.E., and O'Keefe, D.H. Social validation of the headache diary. Behavioral Therapy, 1981, 12, 711-715.

Blanchard, E.B., Andrasik, F., and Silver, B.V. Biofeedback and relaxation in the treatment of tension headaches: a reply to Belar. Journal of Behavioral Medicine, 1980, 3, 227-232.

Blanchard, E.B. and Epstein, L.H. A biofeedback primer. Addison-Wesley Publishing Company, Don Mills, Ontario, 1978.

Blanchard, E.B., Theobald, D.E., Williamson, D.A., Silver, B.V. and Brown, D.A. Temperature biofeedback in the treatment of migraine headaches: a controlled evaluation. Archives of General Psychiatry, 1978, 35, 581-588.

Blanchard, E.B. and Young, T.D. Clinical applications of biofeedback training: a review of evidence. Archives of General Psychiatry, 1974.

Braun, B.G. A case of biofeedback failure. American Journal of Clinical Biofeedback, 1980, 3, 179-180.

Brown, B.B. New Mind, New Body. Bantam Books, New York, 1974.

Brown, B.B. Stress and the Art of Biofeedback. New York: Harper and Row Publishers, 1977.

Budzinski, T.H. Biofeedback strategies in headache treatment. In J.V. Basmajian (Ed.), Biofeedback Principles and Practice for Clinicians. Baltimore: Williams and Williams Company, 1979.

Budzinski, T.H., and Stoyva, J.M. An instrument for producing deep muscle relaxation by means of analog information feedback. Journal of Applied Behavioral Analysis, 1969, 2, 231-237.

Budzinski, T.H., Stoyva, J.M. and Adler, C. Feedback-induced muscle relaxation: applications to tension headache. Journal of Behavioral Therapy and Experimental Psychiatry, 1970, 1, 205-211.

Burchfield, J.R. The stress response: a new perspective. Psychosomatic Medicine, 1979, 41, 661-672.

Campbell, R.J. Psychiatric Dictionary (Fifth Edition) Oxford University Press: New York, 1981.

Carney, P. Psychophysiological Stress, Biofeedback and Migraine Therapy. Unpublished thesis, University of Alberta, 1981.

Check, W.A. Chronic vasoconstriction may have role in migraine headaches. Archives of Internal Medicine, 1980, 140, 19.

Claghorn, J.T., Mathew, R.J., Targen, J.W. and Meyer, J.S. Directional effects of skin temperature self-regulation on regional cerebral blood flow in normal subjects and migraine patients. American Journal of Psychiatry, 1981, 138, 1182-1187.

Cohen, B.A. Basic biofeedback electonics for the clinician. In J.V. Basmajian (Ed.), Biofeedback - Principles and Practice for Clinicians. Baltimore: Williams and Williams Company, 1979

Cohen, M.J., McArthur, D.L. and Rickles, W.H. Comparison of four biofeedback treatments for migraine headache: Psysiological and headache variables. Psychological Medicine, 1980, 42, 463-480.

Cohen, M.J., Rickley, W.H. and McArthur, D.L. Evidence for psychological response stereotypy in migraine headache. Psychosomatic Medicine, 1978, 40, 344-354.

Cott, A., Goldman, J.A., Pavloski, R.P., Kirschberg, G.J. and Fabich, M. The long-term therapeutic significance of the addition of electromyographic biofeedback to relaxation training in the treatment of tension headaches. Behavior Therapy, 1981, 12, 556-559.

Cox, D.J., Freundlich, A. and Meyer, R.G. Differential effectiveness of electromyograph feedback, verbal relaxation instructions, and medication placebo with tension headaches. Journal of Consulting and Clinical Psychology, 1975, 43(6), 892-898.

Cox, J.P., Evans, J.F., and Jamieson, J.L. Aerobic power and tonic heart rate responses to psychosocial stressors. Personality and Social Psychology Bulletin, 1979, 5, 160-163.

Cox, T. Stress. Baltimore: University Park Press, 1978.

Daniels, T.K. The effects of automated hypnosis and hand warning on migraine: a pilot study. The American Journal of Clinical Hypnosis, 1976, 19, 91-94.

Danskin, D.G., and Crow, M.A. Biofeedback: An Introduction and Guide. Mayfield Publishing Company, 1981.

Diamond, S., Diamond-Falk, J., and DeVeno, T. Biofeedback in the treatment of vascular headache. Biofeedback and Self-Regulation, 1978, 3(4), 385-408.

Diamond S. and Medina, J.L. Headaches. Clinical Symposia, 33(2), 1981.

Dudley, R. and Rowland, W. How to find relief from migraine. William Collins, Sons and Company, 1981.

Edmeads, J. Cerebral blood flow in migraine. Headache, 1977, 17, 148-152.

Elder, S.T. and Frentz, K.G. Operant control of surface body temperature. Bulletin of Psychosomatic Society, 1978, 12, 53-54.

Eysenck, H.J. Behavior therapy and the philosophers. Behavior Research and Therapy, 1979, 17, 511-514

Fahrion, S.L. Autogenic biofeedback for migraine. Psychiatric Annals, 1978, 8, 219-234.

Fanchamps, A. The role of humoral mediators in migraine headache. Canadian Journal of Neurological Sciences, 1974, 1, 189-195

Fair, P.L. Biofeedback strategies in psychotherapy. In J.V. Basmajian (Ed.), Biofeedback - Principles and Practice for Clinicians. Baltimore: Williams and Williams Company, 1979.

Fenz, W.D. Strategies for coping with stress . In L.G. Sarason and C.D. Spielberger (Eds.), Stress and Anxiety, (Vol. 2). New York: John Wiley and Sons, 1975, 305-336.

Feuerstein, M. and Adams, H.E. Cephalic vasomotor feedback in the modification of migraine headache. Biofeedback and Self Regulation, 1977, 2, 241-254.

Friar, L.R. and Beatty, J. Migraine: Management by trained control of vasoconstriction. Journal of Consulting and Clinical Psychology, 1976, 44 (1), 46-53.

Friedman, A.P. Prophylaxis of migraine. Headache, 1973, 13, 104-106.

Friedman, A.P. Migraine. Psychiatric Annals, 1975, 5, 271-278.

Fuller, G.D. Current status of biofeedback in clinical practice. American Psychologist, January 1978, 30-48.

Gainer, J.C. Temperature discrimination training in the biofeedback treatment of migraine headache. Journal of Behavior Therapy and Experimental Psychiatry, 1978, 9, 185-188

Garfield, S.L. and Bergin, A.E. Handbook of Psychotherapy and Behavior Change. New York: John Wiley and Sons, 1978.

Gatchel, R.J. and Price, K.P. Clinical Applications of Biofeedback: Appraisal and Status. Toronto: Pergamon Press, 1979.

Gellhorn, E. Principles of autonomic-somatic integrations: Physiological basis and psychological and clinical implications. Minneapolis: University of Minnesota Press, 1976.

Gellhorn, E. and Kiely, W.F. Mystical states of consciousness: Neurophysiological and clinical aspects. The Journal of Nervous and Mental Disease, 1972, 154, 399-405.

Green, E. and Green, A. Beyond Biofeedback. New York: Delacorte, 1977.

Green, E.E. and Green, A. General and specific application of thermal biofeedback. In J.V. Basmajian (Ed.), Biofeedback - Principles and Practice for Clinicians. Baltimore: Williams and Williams Company, 1979.

Green, E.E., Walters, E.D., Green, A.M. and Murphy, G. Feedback techniques for deep relaxation. Psychophysiology, 1969, 6, 371-377.

Grunbaum, A. The placebo concept. Behavior Research and Therapy, 1982, 19, 157-167.

Hanington, E. Migraine. A platelet hypothesis. Biomedicine, 1979, 30, 65-66.

Harris, R.J. A Primer of Multivariate Statistics. New York: Academic Press, 1975.

Hassett, J. A Primer of Psychophysiology. San Francisco: W.H. Freeman and Company, 1978.

Haynes, S.N., Griffin, P., Mooney, D., and Parise, M. Electromyographic biofeedback and relaxation instructions in the treatment of muscle contraction headaches. Behavior Therapy, 1975, 6, 672-678.

Hutchings, D.F. and Reinking, R.H. Tension headaches: What form of therapy is most effective? Biofeedback and Self-Regulation, 1976, 1, 183-190.

Jencks, B. Your Body: Biofeedback at its Best. Chicago: Nelson Hall, 1977.

Johnson, W.G. and Turin, A. Biofeedback treatment of migraine headache: A systematic case study. Behavior Therapy, 1975, 6, 394-397.

Jones, G. Visceral Learning Toward a Science of Self-Control. New York: The Viking Press, 1973.

Katkin, E.S. and Goldband, S. The placebo effect and biofeedback. In R.S. Gatchel and K.P. Price (Eds.), Clinical Applications of Biofeedback: Appraisal and Status. Toronto: Pergamon Press, 1979.

Kazdin, A.E. Non-specific treatment factors in psychotherapy outcome research. Journal of Consulting and Clinical Psychology, 1979, 47, 846-851.

Keefe, F.J. Conditioning changes in differential skin temperature. Perceptual and Motor Skills, 1975, 40, 283-288.

Kewman, D.G. Voluntary control of digital skin temperature for treatment of migraine headaches. Dissertation Abstracts International, 1978, 38, 3399-3400.

Kewman, D.G. and Roberts, A.H. Skin temperature biofeedback and migraine headaches: A double-blind study. Biofeedback and Self-Regulation, 1980, 5, 327-345.

Kimmel, A.M. The relevance of experimental studies to clinical applications of biofeedback. Biofeedback and Self-Regulation, 1981, 6, 263-271.

Kolb, B. and Whishaw, I.Q. Fundamentals of Human Neuropsychology. San Francisco: W.H. Freeman and Company, 1980.

Kudrow, L. Current aspects of migraine headache. Psychosomatics, 1978, 19, 48-57.

Lacey, J.I., Bateman, D.E. and Van Lehn, R. Autonomic response specificity: An experimental study. Psychosomatic Medicine, 1953, 15, 8-21.

Lacey, J.I. and Lacey, B.C. Verification and extension of the principle of autonomic response stereotypy. American Journal of Psychology, 1958, 71, 50-73.

Largen, J.W., Mathew, R.J., Dobbins, K. and Claghorn, J.L. Specific and non-specific effects of skin temperature control in migraine management. Headache, 1981, 21, 36-44.

Libo, L.M. and Arnold, G.E. Continuing relaxation practice after biofeedback therapy: How much is enough? Presented at Biofeedback Society of America Annual Convention, Chicago, Illinois, March, 1982.

Mathew, N.T. Prophylaxis of migraine and mixed headache. Headache, 1980, 20(3), 163-164.

Meen, K. Psychophysiological reactivity in migraine headache. Unpublished Masters thesis, University of Alberta, 1981.

Miller, S.M. Controllability and human stress: Method, evidence and theory. Behavior Research and Therapy, 1979, 17, 287-304.

Mitchell, K.R. and White, R.G. Behavioral self-management: An application to the problem of migraine headaches. Behavior Therapy, 1977, 8, 213-221.

Morrison, D.F. Multivariate Statistical Methods. New York: McGraw-Hill, 1967.

Orne, M.T. The efficiency of biofeedback therapy. Annual Revised Medicine, 1979, 30, 489-503.

Ovilansky, A. et al. Release of platelet 5-hydroxytryptamine by plasma taken from patients during and between migraine attacks. Pain, 1976, 2, 315-318.

Payne, C.M. Biofeedback training in digital vasomotor control in migraineurs and normal subjects. Dissertation Abstracts International, 1979, 40, 460-461.

Pearce, J. Migraine: A psychosomatic disorder. Headache, 1977, 17, 125-128.

Price, K.P. Biofeedback and migraine. In R.J. Gatchel and K.P. Price (Eds.) Clinical Applications of Biofeedback: Appraisal and Status. Toronto: Pergamon Press, 1979.

Qualls, P.J. and Sheehan, P.W. Electromyograph feedback as a relaxation technique: A critical appraisal and reassessment. Psychological Bulletin, 1981, 90, 21-42.

Reading, C. and Mohr, P.D. Biofeedback control of migraine: A pilot study. British Journal of Social and Clinical Psychology, 1976, 15, 429-433.

Reinking, R.H. and Kohl, M.L. Effects of various forms of relaxation training on physiological and self-report measures of relaxation. Journal of Consulting and Clinical Psychology, 1975, 43(5), 595-600.

Sakellaris, P.C. and Vernikos-Danellis, J. Increased rate of response of the pituitary-adrenal system in rates adapted to chronic stress. Endocrinology, 1975, 97, 597-602.

Saper, J.R. Migraine: Treatment. Journal of the American Medical Association, 1978, 239(23), 2480-2484.

Saper, J.R. and Mayee, K.R. Freedom from headaches. New York: Simon and Schuster, 1978.

Sargent, J.D. Head, Migraine Project, The Menninger Foundation, Topeka K.S. 1979. Personal Communication in D.G. Danskin and M.A. Crow. Biofeedback: An Introduction and Guide. Mayfield Publishing Company, 1981.

Sargent, J.D., Coyne, L. and Solback, P. Design and implementation of a controlled evaluation of biofeedback as a treatment for migraine headaches. American Journal of Clinical Biofeedback, 1978, 1, 1-9.

Sargent, J.D., Green, E.E. and Walters, E.D. The use of autogenic feedback training in a pilot study of migraine and tension headache. Headache, 1972, 12, 120-125.

Sargent, J.D., Green, E.E. and Walters, E.D. Preliminary report on the use of autogenic feedback training in the treatment of migraine and tension headaches. Psychosomatic Medicine, 1973, 35(2), 129-135.

Sargent, J.D., Walters, E.D. and Green, E.E. Psychosomatic self-regulation of migraine headaches. Seminars in Psychiatry, 1973, 5, 415-428.

Scheife, R.T. and Hills, J.R. Migraine headache: Signs and symptoms, biochemistry and current therapy. Drug Therapy Reviews: American Journal of Hospital Pharmacy, 1980, 37(3), 365-374.

Schnarch, D.M. and Hunter, J.E. Migraine incidence in clinical vs non-clinical populations. Psychosomatics, 1980, 21(4), 314 -325.

Schrader, S.L. A comparison of two biofeedback treatments for migraine headache. Dissertation Abstracts International, 1979, 40, 1913

Schwartz, G.E. Psychological foundations of psychotherapy and behavior change. In S.L. Garfield and A.E. Bergin (Eds.) Handbook of Psychotherapy and Behavior Change. New York: John Wiley and Sons, 1978.

Schwartz, G.E. and Beatty, J. Biofeedback: Theory and Research. New York: Academic Press, 1977.

Selby, G. and Lance, J,W. Observations on 500 cases of migraine and allied vascular headache. Journal of Neurological and Neuropsychological Psychiatry, 1960, 23, 23-32.

Sellick, M. Self-esteem, aerobic capacity, and psychosocial stress. Unpublished Bachelor thesis, Lakehead University, 1977.

Sellick, S. Aerobic power and responses to psychosocial stressors. Unpublished Masters thesis, Lakehead University, 1979.

Selye, H. The general adaptation syndrome and the diseases of adaptation. Journal of Clinical Endocrinology, 1946, 6, 117-230.

Selye, H. Stress Without Distress. Scarborought, New American Library, Signet Book, 1974.

Selye, H. The Stress of Life. New York: McGraw-Hill, 1976.

Selye, H. The Stress of My Life. McClelland and Stewart Ltd. Toronto, 1977.

Shapiro, A.K. and Morris, L.A. Placebo effects in medical and psychological therapies. In S.L. Garfield and A.E. Bergin (Eds.). Handbook of Psychotherapy and Behavior Change. New York: John Wiley and Sons, 1978.

Sheldon, J.J. Blood Vessels of the Scalp and Brain. Clinical Symposia, 1981, 33(5).

POOR COPY
COPIE DE QUALITEE INFERIEURE

Silver, B.V., Blanchard, E.B., Williams, D.A., Theobald, D.E. and Brown, D.A. Temperature biofeedback and relaxation training in the treatment of migraine headaches: One-year follow-up. Biofeedback and Self-Regulation, 1979, 4, 359-366.

Sloan, P.E. A comparison of the effectiveness of autogenic feedback training vs electromyogram feedback assisted relaxation for the treatment of migraine headaches. Dissertation Abstracts International, 1978, 38, 5045.

Sovak, M., Kunzel, M., Sternback, R.A. and Dalessio, D.J. Is volitional manipulation of hemodyamics a valid rationale for biofeedback therapy of migraine? Headache, 1978, 18, 197-202.

Sovak, M., Kunzel, M., Sternback, R.A. and Dalessio, D.J. Mechanism of the biofeedback therapy of migraine: volitioral manipulation of the psychophysiological background. Headache, 1981, 21, 89-92.

Spierings, E.L.H. Serotonin and its inhibitors. Headache, 1980, 20, 149.

Spierings, E.L.H. and Saxena, P.R. The action of ergotamine on the distribution of carotid blood flow - the migraine shunt theory revisited. Headache, 1980, 20, 143-145.

Stoyva, J. Guidelines in the training of general relaxation. In J. Basmajian (Ed.), Biofeedback - Principles and Practice for Clinicians. Baltimore: Waverly, 1979.

Stroebel, C.F. and Glueck, B.C. Biofeedback treatment in medicine and psychiatry: An ultimate Placebo? Seminars in Psychiatry, 1973, 5, 378-393.

Sturgis, E.T. Lability and reactivity in the headache response. Dissertation Abstracts International, 1980, 40, 3425-3426.

Surwit, R.S. and Keefe, F.J. Frontalis EMG feedback training: An electronic panacea? Behavior Therapy, 1978, 9, 779-792.

Surwit, R.S., Shapiro, D. and Feld, J.L. Digital temperature autoregulation and associated cardiovascular changes. Psychophysiology, 1976, 13, 242-248.

Taub, E. Self-regulation of human tissue temperature. In G.E. Schwartz and J. Beatty. Biofeedback: Theory and Research. New York: Academic Press, 1977.

POOR COPY
COPIE DE QUALITEE INFERIEURE

Taub, E. and Emurian, C.S. Feedback - aided self-regulation of skin-temperature with a single feedback locus. Biofeedback and Self-Regulation, 1976, 1(2), 147-168.

Taub, E. and Stroebel, C.F. Biofeedback in the treatment of vasoconstrictive syndromes. Biofeedback and Self-Regulation, 1978, 3(4), 363-373.

Thompson, C.S. Autogenic feedback training: The effects of outcome and accessibility of hand temperature biofeedback on the reduction of migraine headaches. Dissertation Abstracts International, 1977, 37, 3635-3636.

Thompson, J.K. Haber, J.D. and Tearnan, B.H. Generalization of frontalis electromyographic feedback to adjacent muscle groups: A critical review. Psychosomatic Medicine, 1981, 43, 19-24.

Tryon, W.W. A methodological critique of Bandura's self-efficacy theory of behavior change. Journal of Behavior Therapy and Experimental Psychiatry, 1981, 12(2), 113-114.

Turin, A. and Johnson, W.G. Biofeedback therapy for migraine headaches. Archives of General Psychiatry, 1976, 33, 517-519.

Waters, W.E. and O'Connor, P.J. Prevalence of migraine. Journal of Neurology, Neurosurgery and Psychiatry, 1975, 38, 613-616.

Weiss, T. Weaning in biofeedback training. American Journal of Psychiatry, 1975, 132, 1220.

Weiss, T. and Engel, B.T. Operant conditioning of heart rate in patients with premature ventricular contractions. Psychosomatic Medicine, 1971, 33, 301-321.

Wickramasekera, I, A conditioned response model of the placebo effect: Predictions from the model. Biofeedback and Self-Regulation, 1980, 5(1), 5-15.

Wilkins, W. Expectancies in therapy research: Discriminating among heterogeneous nonspecifics. Journal of Consulting and Clinical Psychology, 1979, 47, 837-845.

Williams, R.B. (Jr.) and Gentry, W.D. (Eds.) Behavioral Approaches to Medical Treatment. Cambridge, Mass.: Ballinger Publishing Co., 1977.

Winer, B.J. Statistical Principles in Experimental Designs. McGraw -Hill, 1962.

Wittrock, M.C. The Human Brain. S. Englewood Cliffo: Prentice-Hall, Inc., 1977.

Wolpe, J. Reciprocal inhibition and therapeutic change. Journal of Behavior Therapy and Experimental Psychiatry, 1981, 12, 185-188.

Yates, A.J. Biofeedback and the Modification of Behavior. New York: Plenum Press, 1980.

APPENDICES

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APPENDIX A

CRITERION FOR INCLUSION--TELEPHONE INTERVIEW

Name:_____ Address:_____

Phone:_____ Sex: _____ Postal Code:_____

INCLUSION CRITERIA:	Criteria
1. What is your age?	_____ (18 to 55)
2. How often have your headaches occurred in the last 2 months	_____ (less than 3X per day & ≥ 1 per month)
3.(a) Do you take any medication for your headache?	_____
(b) What is the name of the medication?	_____
(c) How well does it control headache?	_____(No)
(d) Currently using oral contaceptives?	_____(No); pregnant? _____(No)
(e) Are you on any other medications? specify:	_____
4. Have your headaches occurred one or more times per month over the past 2 years?	_____ (Yes)
5. Are you currently receiving any form of psychotherapy?	_____(No)
6. Do you ever experience sensory losses or paralysis of some muscles during a headache?	_____ (No)
7. Do you suffer from a convulsive disorder (epileptic seizures)?	_____ (No)
8. Do you have any form of heart disease or disorder?	_____ (No)
9. Do you have any health problem such as diabetes, hypertension, etc? specify:	_____

SUBJECT MUST REPORT "YES" TO THREE (3) OF THE FOLLOWING FIVE (5) ITEMS:

_____A. Does the head pain sometimes exist on one side of the head only?

_____B. Is the head pain generally pulsative (or throbbing)?

_____C. Does nausea or vomiting generally accompany the headache?

_____D. Does sensitivity to light generally accompany the headache?

_____E. Has your headache been diagnosed as a migraine by your physician?

Available times for treatment:

(1st and 2nd choice)

	Mon/Thurs.	Tues/Fri	Wed/Sat
Morning	:	:	
Afternoon	:	:	
Evening	:	:	

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APPENDIX B

TREATMENT CONTRACT

Participant - I understand that my participation in the migraine treatment program will require my full cooperation in each of the following components of the study:

1) Punctual attendance at all treatment sessions scheduled;

2) Attendance at one pre-training orientation session held several days before treatment and one post-treatment follow-up session held one month after the treatment period;

3) One half-hour daily practice of specific relaxation skills learned in treatment to continue throughout the training period;

4) Hourly monitoring of headache activity and medication consumption prior to and throughout the training period;

5) Keeping a headache diary for one year following the treatment period;

6) Notifying your counsellor by phoning 432-5214 (during regular office hours) if unable to keep a scheduled appointment.

7) Notifying the Department of Educational Psychology, 6th floor - Education North, University of Alberta should my address change.

Date:_____ Signature:_____

Counsellor - I promise that all records of Participants' names, addresses and personal information will be kept confidential. At the completion of this study a summary of the results obtained shall be made available to all those who fully participated.

Date:_____ Signature:_____

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APPENDIX C

MEDICAL FORM

Name of Physician: _____

Name of Patient: _____

Date of Birth: _____

Address: _____

_____

Phone: _____

The above named patient has been selected to participate in a treatment program for migraine patients being conducted at the University of Alberta, Department of Educational Psychology. This research is being supervised by Dr. George Fitzsimmons. The techniques being used may include relaxation training and psychophysiological monitoring including electromyography, galvanic skin response, and surface skin temperature.

We are requesting each patient to obtain the signature of their physician to verify that they have received a recent medical examination and to ensure that there is no medical reason why they should not participate in the research project.

For Physician

(A) This is to certify that _____ has been medically examined and I do not advise against his/her participation in the program described.

(B) I _____ (do, do not) agree that the headache pain which this person reports is of the migraine form.

Date: _____

Physician's Signature: _____

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APPENDIX D

HOURLY RECORD FORM

PROGRESS CHART

It is important to monitor the intensity of your headaches for at least two reasons:

1. Research has shown that this will help to reduce the psychological side affects that often accompany a headache.

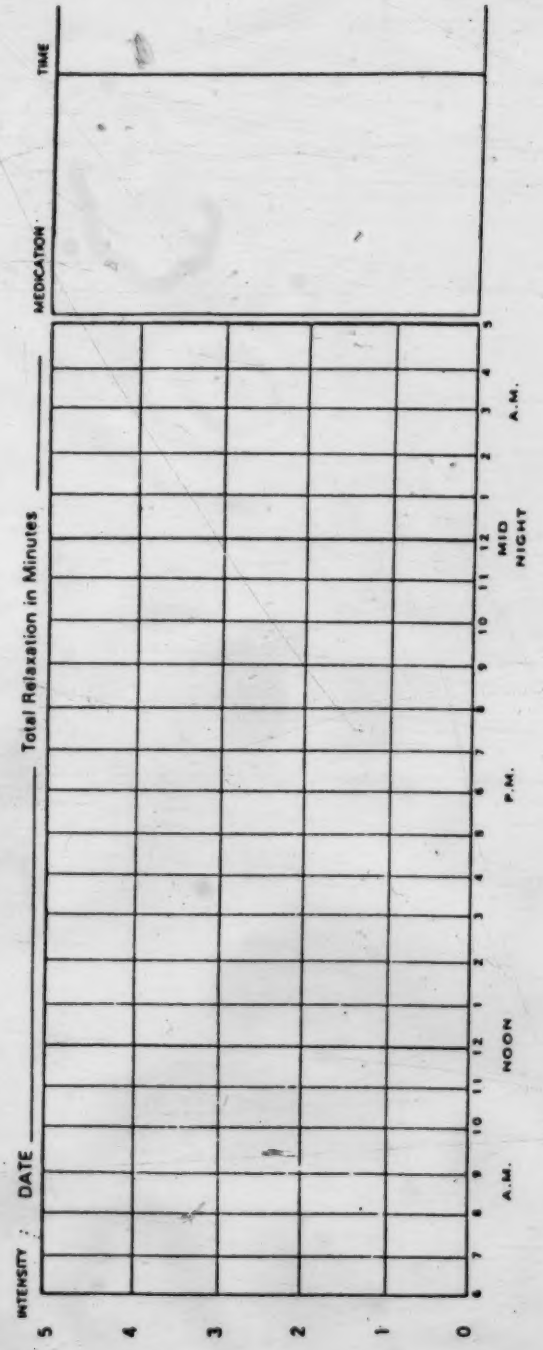
2. It is useful in helping to determine the affects of your treatment program.

The following five point scale is useful in helping people monitor the severity of their headache.

0 — No headache.
1. — Low level, only enters awareness when you think about it.
2 — Aware of headache most of the time but it can be ignored at times.
3 — Painful headaches but still able to continue job.
4 — Severe headache, difficult to concentrate with undemanding tasks.
5 — Intense incapacitating headache.

To monitor your headache level mark the appropriate number on the graph at each hour and join the points together. Placing the coloured dot on your watch will help you remember to do this.

NAME: _____



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APPENDIX E

DAILY RECORD FORM

DAILY RECORD: MIGRAINE HEADACHE PATTERN

NAME: _____

MONTH: _____

This record is to be completed at the end of each day.

As we mentioned to you at the beginning of this program, the only information that we will be able to obtain regarding long-term benefits of biofeedback requires your continued assistance. This is a daily record. What we require of you are the following:

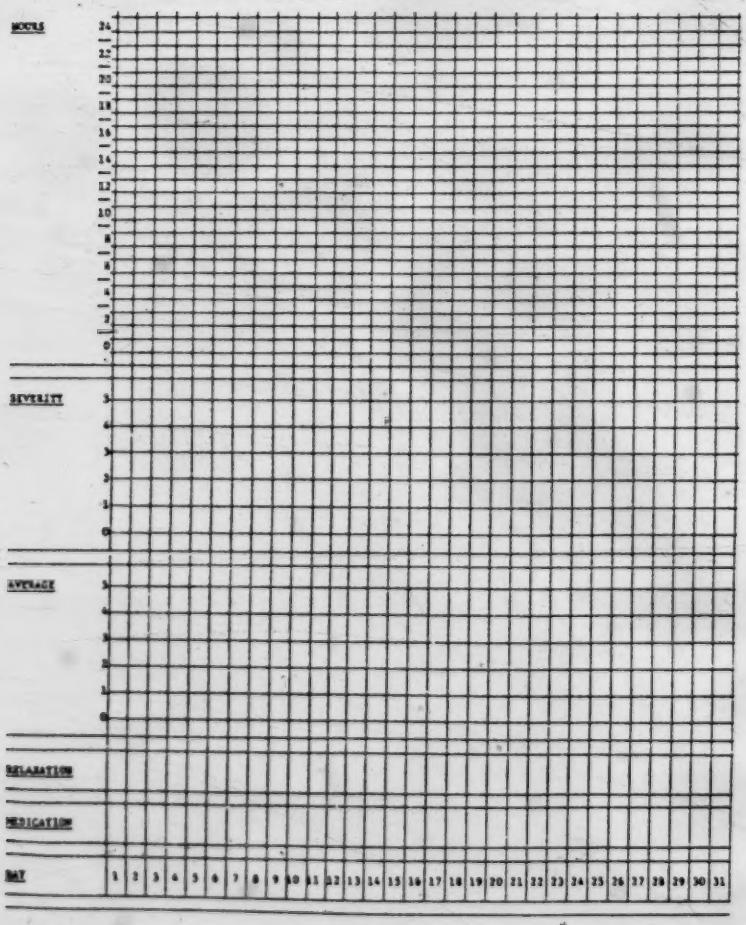
A) Record the number of hours that you had a migraine headache during the day.

B) Record the highest degree of severity experienced during that day.

C) Record the average degree of severity experienced during that day.

D) Record the number of minutes spent on relaxation (of the nature introduced to you during our treatment phase).

E) Record medication taken that day for the relief of migraine headache pain (name and amount in milligrams).



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APPENDIX F

PSYCHOPHYSIOLOGICAL PROFILE PROCEDURE

1. Record room temperature. If not 71-73 degree Fahreheit, reschedule.

2. Seat client in recliner chair and tilt chair to the first recliner position. Inquire whether a headache is now present and if so, reschedule the session.

3. Hook-Up: EMG--frontal area with electrodes spaced one inch apart; Temperature--non-dominant hand.

4. Explanation: read the following to the client:

> Today's session will last approximately forty minutes. What I am going to do is attach you to two biofeedback instruments in order to see what levels of activity you produce in two different physiological systems, skin temperature, and muscle tension. These instruments will not shock you or harm you in any way, they are merely attached onto the surface of your skin with these wires. We are hooking you up today in order to find out how your body activity corresponds to your headache pattern, and how the relaxation treatment program changes both your body activity and your headache pattern. Do you have any questions?

5. Relaxation Instructions: read the following to the client:

> For the next five minutes I would like you to relax comfortably with your eyes open and just listen to the music being played in the background. Try to avoid unpleasant thoughts and just enjoy this five minutes of rest. After five minutes have elapsed I will ask you to sit for fifteen minutes with your eyes closed. Please sit quietly without moving or talking and keep your hands on the arm rest with your palms facing upward. Do you have any questions? Okay then, starting with your eyes open just relax and listen to the music that will be playing. I will tell you when five minutes are up.

6. Turn on EMG, temperature, optical isolators, computer, and printer.

7. Settings: EMG scale @ 1., temperature @ 10.

8. Identify client on ticker tape (name, date, therapist).

9. Turn on music--low volume.

10. Start computer and start timing five minutes of adaptation. After five minutes draw a line on the ticker tape and indicate end of phase number one.

11. Relaxation period: read the following to the client:

> Now I would like you to sit for fifteen minutes with your eyes closed. Continue listening to the music and try not to fall asleep.

Start timing fifteen minutes of relaxation.

12. Stress period: read the following to the client:

> Okay, while keeping your eyes closed I want you to perform a mental task for me. I want you to subtract seven from 1000 and then to continue subtracting seven from your answer as fast as possible until I tell you to stop. Then I will ask you what number you got to. Do this in your head, not out loud. So, 1000 minus seven is . . . and keep going. (no music)

After three minutes say "stop," and ask how far the subject was able to subtract.

13. Recovery period: read the following to the client:

> Now I want you to relax again with your eyes closed and listen to the music without interruption for five minutes, and then we are finished for today.

After five minutes end the session and disconnect equipment.

APPENDIX G

E.M.G. Training Rationale

The treatment sessions you are receiving are designed to teach you how to produce more effective physiological relaxation at will. Your final goal in treatment is to become able to discriminate excessive stress in your body and be able to remove such stress in order to prevent migraine headaches. Regular and consistent practice at removing excessive stress will eventually develop into a life-style habit. When this occurs your body will maintain a more relaxed level of arousal without conscious effort. It may take somewhere between a couple of weeks to several months to develop this automatic habit, depending upon the amount of relaxation practice you do and the strength of the stress habit you now have.

In biofeedback training you will learn to relax efficiently, guided by the feedback signal. The idea is to decrease your muscle tension voluntarily as you relax and learn to use decreased muscle tension as an index of your relaxation level. Over time you will learn to produce greater levels of relaxation in less time and to maintain these levels for longer periods. Even though the biofeedback is only attached to the head region it is to your advantage to learn to decrease your muscle tension as part of a total body relaxation response.

Biofeedback guided relaxation takes place in three stages. The first stage is called the "awareness" stage where you brain is merely made aware of how much feedback corresponds to how much muscles tension. Gradually the second stage emerges where in addition to becoming aware of tension levels you become able to control the tension and further reduce it. This second stage is known as the "control" stage.

Please note that the control stage takes time to emerge because you must learn the skill involved. Also note that contrary to most other intentional learning you do, learning to relax does not involve active striving. The more you strive the more tense you will become. Instead of actively striving to reduce muscle tension you must passivily concentrate on the feedback signal and "allow" the tension to reduce. In other words, "let it happen".

The final stage of biofeedback guided relaxation, following awareness and control is the "weaning" stage. Weaning involves practice at producing the relaxation response in the absence of the biofeedback signal. Such practice will begin once you have learned the relaxation response. In this way you can learn an effective relaxation skill which is not dependent upon biofeedback.

Many persons have asked what thinking strategies they should be using to decrease muscle tension as they passively concentrate. Other than advising such persons to avoid unpleasant thoughts or stress-related rumination there is no particular strategy that everyone will find effective. Some people use mental images of relaxing settings such as laying on a warm beach, skiing down a mountain in slow motion, or watching a beautiful sunset. Others think suggestive phrases to themselves such as "I am becoming warm and relaxed". Others do not think about anything, they let their minds go blank. Most people find some particular strategy useful at first but as they learn to relax efficiently, letting go of tension becomes a skill they can utilize without any conscious strategy. Over the course of the training sessions, I would like you to use whatever strategies you feel comfortable with to relax. But remember, the important thing is not to force any approach or to try too hard, because effort is the opposite of relaxation. Just let the approach you choose flow, just imagine it is already happening.

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APPENDIX H

EMG TRAINING PROCEDURES AND INSTRUCTIONS

1. Check tape in printer.

2. Take room temperature. If not 71-73 degrees Fahreheit, reschedule.

3. Collect forms from client.

4. Mark treatment session number in progress file and on print-out.

5. Attach EMG electrodes and check for allowable impedence levels. (Less than 1.5 microvolts with scale set at X30; if not, re-do).

   Attach temperature thermistor to the palmer surface of the first phalange of the middle finger on the nondominant hand.

6. Check to make sure client has read the rationale (EMG).

7. Check to make sure the biofeedback equipment is functional. Explain EMG needle-guage and make sure other biofeedback information (temperature) is not in view of client.

8. Turn on EMG, temperature, optical isolators, computer, and printer. Turn off flourescent light leaving on the incandescent lamps.

9. Identify client on ticker tape (name, date, session number, therapist.

10. Adaptation period: read the following to the client:

    This session will last approximately forty minutes. Please keep your eyes open during the entire session. The session will consist of four phases. You will not receive any biofeedback during the first three phases. The first phase is an adaptation phase. For the next five minutes just sit in the chair with your eyes open.

11. Start computer and begin timing five minutes.

12. After five minutes draw a line on the printout and say:

    For the next two minutes I will be collecting baseline data. Please continue to sit quietly without talking and continue with your eyes open.

13. After two minutes draw a line on the printout and say:

> For the next two minutes I would like you to decrease your muscle tension.

14. After two minutes draw a line on the printout and say:

> This is the training phase. You will have three periods of five minutes of practice, separated by two one-minute rests. (Uncover the EMG needle guage). As you decrease the muscle tension in your head region the clicking in these headphones will slow down. For the next five minutes I would like to to attend to the needle you can see here and to the clicking you can hear, and practise decreasing your muscle tension.

15. Drawa line on the printout and begin timing five minutes.

16. After five minutes draw a line on the printout and say:

> Please stop practising now and just take a break.

After a one-minute break where you can discuss performance, draw a line on the printout and say:

> Now please begin practising again for five minutes.

Follow with a one-minute rest.

17. Repeat with a third and final five-minute practise session.

18. Conduct written summary procedure. At the end of each session discontinue the biofeedback monitoring and give the following instructions:

> Please take a few minutes and write down a description of the strategies which you employed to relax and also identify any feelings or sensations which appeared to be associated with the slower clicking or the dropping of the needle. A new summary will be written each session to help you solidify in your own minds what strategies were in fact useful to you, and to inform us as to the tactics used.

19. Discuss progress. At the end of each session show each subject the EMG levels they achieved and compare these to the two-minute EMG average value computed at the end of the fifteen minutes of relaxation during the pre-treatment session. Point out that ideally they will be learning to become more relaxed faster, and be able to maintain such relaxed levels longer.

20. Discuss home practice and medication. Encourage subjects to continue home practice for thirty minutes each day and emphasize the importance of this component of the total treatment package. Also ask that each subject continue to carefully monitor their medication intake and to consult with their physician about any changes required in their prescriptions. Inform subjects that increased relaxation may alter the effects of their medication, migraine or otherwise. This is especially true for subjects taking medication for hypertension or diabetes.

APPENDIX I

Temperature Training Rationale

The treatment sessions you are receiving are designed to teach you how to produce more effective physiological relaxation at will. Your final goal in treatment is to become able to discriminate excessive stress in your body and be able to remove such stress in order to prevent migraine headaches. Regular and consistent practice at removing excessive stress will eventually develop into a life-style habit. When this occurs your body will maintain a more relaxed level of arousal without conscious effort. It may take somewhere between a couple of weeks to several months to develop this automatic habit, depending upon the amount of relaxation practice you do and the strength of the stress habit you now have.

In biofeedback training you will learn to relax effeciently, guided by the feedback signal. The idea is to warm your hands voluntarily as you relax and learn how to use hand warming as an index of your relaxation level. Over time you will learn how to produce greater levels of relaxation in less time and to maintain these levels for longer periods. Even though the biofeedback is only attached to one of your hands it is to your advantage to learn how to hand warm as part of a total body relaxation response.

Biofeedback guided relaxation takes place in three stages. The first stage is called the "awareness" stage where your brain is merely made aware of how temperature changes correspond to vascular changes brought about by stress and relaxation. Gradually the second stage emerges where in addition to becoming aware of stress levels you become able to control the stress and further reduce it. This second stage is known as the "control" stage.

Please note that the control stage takes time to emerge because you must learn the skill involved. Also note that contrary to most other intentional learning you do, learning to relax does not involve active striving. The more you strive the more tense you will become. Instead of actively striving to warm your hands you must passively concentrate on the feedback signal and "allow" the warming to occur. In other words, "let it happen".

The final stage of biofeedback guided relaxation awareness and control is the "weaning" stage. Weaning involves practice at producing the relaxation response in the absence of the biofeedback signal. Such practice will begin once you have learned the relaxation response. In this way you can learn an effective relaxation skill which is not dependent upon biofeedback.

Many persons have asked what thinking strategies they should be using to induce hand warming as they passively concentrate. Other than advising such persons to avoid unpleasant thoughts or stress-related ruminations there is no particular strategy that everyone will find effective. Some people use mental images of relaxing settings such as laying on a warm beach, skiing down a mountain in slow motion, or watching a beautiful sunset. Others think suggestive phrases to themselves such as "I am becoming warm and relaxed". Others do not think about anything, they let their minds go blank. Most people find some particular strategy useful at first but as they learn to relax efficiently, letting go of tension becomes a skill they can utilize without any conscious strategy. Over the course of the training sessions, I would like you to use whatever strategies you feel comfortable with to relax. But remember, the important thing is not to force any approach or to try too hard, because effort is the opposite of relaxation. Just let the approach you choose flow, just imagine it is already happening.

FINE PRINT

APPENDIX J

TEMPERATURE TRAINING PROCEDURES AND INSTRUCTIONS

1. Check tape in printer.

2. Take room temperature. If not 71 - 73 degrees Fahrenheit, reschedule.

3. Collect forms from client.

4. Mark treatment session number in progress file and on print-out.

5. Attach EMG electrodes and check for allowable impedence levels. (Less than 1.5 microvolts with scale set at X30; if not, re-do).

   Attach temperature thermistor to the palmer surface of the first phalange of the middle finger on the nondominant hand.

6. Check to make sure client has read the rationale (temperature).

7. Check to make sure the biofeedback equipment is functional. Explain temperature needle-guage and make sure other biofeedback information (EMG) is not in view of client.

8. Turn on EMG, temperature, optical isolators, computer, and printer. Turn off flourescent light leaving on the incandescent lamps.

9. Identify client on ticker tape (name, date, session number, therapist).

10. Adaptation period: read the following to the client:

    This session will last approximately forty minutes. Please keep your eyes open during the entire session. The session will consist of four phases. You will not receive any feedback during the first three phases. The first phase is an adaptation phase. For the next five minutes just sit in the chair with your eyes open.

11. Start computer and begin timing five minutes.

12. After five minutes draw a line on the printout and say:

    For the next two minutes I will be collecting baseline data. Please continue to sit quietly without talking and continue with your eyes open.

13. After two minutes draw a line on the printout and say:

    For the next two minutes I would like you to increase your skin temperature.

14. After two minutes draw a line on the printout and say:

    This is the training phase. You will have three periods of five minutes of practice, separated by two one-minute rests. (Uncover the temperature needle-guage). As you increase the temperature in your hands the needle you see here will indicate that change and the tone you will hear through the headphones will also change. If you are able to change your skin temperature by two degrees, just try and maintain that change for the duration of the five minute practice.

    If the subject is presently below ninety degrees, begin by warming and placing an arrow above the needle-guage to indicate the direction that the client will be attempting to move the needle. If the client is above ninety degrees, begin by cooling, and placing the arrow in the appropriate direction.

15. Draw a line on the printout and begin timing five minutes.

16. After five minutes draw a line on the printout and say:

    Please stop practising now and just take a break.

    After a one-minute break where you can discuss performance, draw a line on the printout and say:

    Now please begin practising again for five minutes.

    If the subject is below ninety degrees, continue to have the subject warm. If the subject is above ninety degrees, change direction of arrow and have the subject cool for the five minutes.

    Follow with a one-minute rest.

17. Repeat with a third and final five-minute practise session. If the subject is below ninety degrees, continue warming. If the subject has been cooling, warm regardless of temperature.

18. Conduct written summary procedure. At the end of each session discontinue the biofeedback monitoring and give the following instructions:

Please take a few minutes and write down a description of the strategies which you employed to relax and also identify any feelings or sensations which appeared to be associated with the warming and cooling of your hands. A new summary will be written each session to help you solidify in your own mind what strategies were in fact useful to you, and to inform us as to the tactics used.

19. Discuss progress. At the end of each session show each subject the temperatures they achieved and compare these to the temperature levels at various times during the adaptation phase and the no-feedback temperature increasing phase. Point out that ideally they will be learning to become more relaxed faster, and will be able to maintain such relaxed levels longer.

20. Discuss the importance of home practice, using the audio cassette tapes, and the daily monitoring of medication intake. Emphasize the importance of daily relaxation as being a very important component of the total treatment package. Also ask that each subject continue to carefully monitor their medication intake and to consult with their physician about any changes required in their prescriptions. Inform subjects that increased relaxation may alter the effects of their medication, migraine or otherwise. This is especially true for subjects taking medication for hypertension or diabetes.

APPENDIX K

Biotic Band Monitoring and Recording

Please use your Biotic Band device to monitor finger temperature daily while you listen to the relaxation tape. Attach the band to the middle finger of your non-dominant hand. Place the band with the temperature scale on the palmer surface of your finger and center it mid-way along the length of your finger. The band should be snug but not tight. While relaxing try to sit in a comfortable chair with arm rests so that your hand temperature will not be effected by warmth from your lap.

As you practise relaxation note how your finger temperature increases. On your headache monitoring form, write down your finger temperature: (a) after the band has been on your finger for 1 minute and before you start the tape, and (b) at the end of the tape.

Please avoid crushing or crumpling the band as they may become inaccurate with abuse. If you think that your band has broken bring it in to your next training session. The bands must be returned at the end of treatment.

BIOTIC-BAND II has a range of 20.0 F divided into two degree intervals which are indicated on the band by the printed numbers. The liquid crystal squares beside the numbers light up when the temperature of the finger being monitored comes within that two degree range. Within each range of two degrees, color changes indicate smaller changes in the temperature. Each color change equals a change of 0.5 F as shown in the table below.

Lighted Degree	Red-Tan	Orange	Yellow-Green	Blue-Green	Blue
78	78	78.5	79	79.5	80
80	80	80.5	81	81.5	82
82	82	82.5	83	83.5	84
84	84	84.5	85	85.5	86
86	86	86.5	87	87.5	88
88	88	88.5	89	89.5	90
90	90	90.5	91	91.5	92
92	92	92.5	93	93.5	94
94	94	94.5	95	95.5	96
96	96	96.5	97	97.5	98

In taking a reading always read the highest temperature showing. The purple color which may sometimes be visible on some squares should always be ignored.

APPENDIX L

CORRESPONDENCE DURING YEAR OF FOLLOW-UP

Summer 1981

We wish to thank each and every one of you for your involvement and assistance in this project, we couldn't have done it without you! It is our hope that this has been a worthwhile endeavour for you as well.

Should you need to contact me concerning change of address or significant changes in your headache pattern (or anything else related to this study for that matter), please call me at 464-5470 (evenings), or at the university until the end of August at 432-5205.

Again, thank you very much . . . I will be in touch with you in about 2 months.

Scott Sellick.

SEPTEMBER 15, 1981

TO ALL PARTICIPANTS OF THE MIGRAINE HEADACHE--BIOFEEDBACK STUDY:

Well hello there one and all! Let me first of all thank each one of you for your continued support and encouragement as I carry on this research for a few more months. As partial payment for your assistance I am seriously considering inviting each of you who are able to maintain contact with me to return for a "booster session" once I have received sufficient follow-up information.

Please find enclosed: 1] an envelope for you to return completed record sheets,
2] four more months worth of recording sheets,
3] a little bit of information about what Marnie and Penny found out as they completed their Masters degrees.

I cannot emphasize enough how IMPORTANT this follow-up data is to the continuing evaluation of biofeedback and migraine headaches. You see, I need this information in order to complete the work I have begun. Without this follow-up, for me, the initial five-months of work will have been in vain. I am now working full-time (interning) and am having to continue my research in the evening and on weekends (violins please). In other words, please attend to this today and continue to take those daily 10 seconds to fill in the information. I really don't have the time to call and remind ("bug") you but I will be forced to if I don't receive your envelopes by October 15.

Thank you all, very much. I often think of you and wonder how your persistence is paying off. Until next time then (early 1982).

Scott Sellick, Psychologist
Hard-working doctoral student . . . . . Scott

The latest from Marnie and Penny is quite encouraging . . . here's hoping that Scott's results are just as exciting as we go along!

Marnie looked specifically at headache activity (severity and duration over a period of time) and discovered that the treatment package was effective for just about everyone. Almost (but not quite) half of you had significant reduction in headache activity and almost (but not quite) three-quaters of you had significant or 'some' improvement the way Marnie looked at things.

Penny looked at the notion that some types of people would respond better to biofeedback than would others. For example, she thought that people who are more prone to think of themselves as being in control of their lives, would benefit more from biofeedback than would people who generally felt like they were tossed to and fro. She found that both kinds of people were capable of being equally competent at biofeedback. That's kind of good news . . . it means you don't have to have a certain 'personality' in order to master biofeedback techniques . . . at least on the type of personality tests she had you complete. Remember those crazy designs you had to find in amongst the other lines? Well, again, those of you who did poorly (and we don't know who you are) did just as well at the biofeedback training as did those of you who did well (and we don't know who you are either). So, while Penny was hoping to be able to predict who would be good at biofeedback and who would not be able to catch on . . . she only found that everyone was equally capable. Sorry Penny, but that's actually good news for migraine sufferers!

January 1982

To all participants of the migraine headache and biofeedback program . . .

First of all let me again express my great appreciation to each one of you who took the time to send in the last set of data and who have continued to fill in those "blessed squares" each day. This project represents the success of my doctoral program (my life), and it looks like most of you have realized its importance to me. I thank you, especially those of you who have not been having such a good time with your headaches recently.

Please find enclosed an envelope to use in returning your completed forms. If you've forgotten to do these charts for a month or so, please go back and fill them out in as accurate a fashion as possible; in a representative manner.

Also, please fill in your address and phone numbers so I'll have accurate information . . . I'll be calling everyone before the middle of February.

Thanks again, I'll be talking to you soon. Please feel free to call me in the mean-time at 464-5470 (evenings--M, W, Th).

Scott Sellick

POOR COPY
COPIE DE QUALITEE INFERIEURE

May 19, 1982.

Hello there one and all . . . . . .

The day has finally come when you receive a letter that says that you will not have to complete forms forever. May 31 is the last day for headache recording and I forbid you to fill in any more of the 'blessed' forms (unless you want to for your own information). So, as of June 1, NO MORE FORMS!

However, one last favour is requested. I am in a very tight position right now with a very strict schedule if I want to make fall convocation. I desperately need you to send this last information back to me before June 8 (my birthday), so that I can get your data into the computer and analyze it before the middle of July. The whole Sellick family is counting on you! (I think I've said enough . . . I'm not interested in making anyone feel guilty for being slow, just asking that you be prompt and help keep me motivated).

I've enclosed an envelope WITH STAMP! If you don't have all the information I need we can talk about it when I call. I will be calling between 7 and 10 (at night) during the first week of June. If I don't get you please call me at work (973-2392, Alberta Hospital) or at home (464-5470).

Thank you all, VERY much. I will be forever grateful to each one of you! I will be in touch . . . . . .

Scott Sellick

POOR COPY
COPIE DE QUALITEE INFERIEURE

APPENDIX M

Characteristics of Study Population

at End of Treatment Phase

		Percentage of Respondents--
1.	Able to tell that a migraine is coming before the headache actually begin (prodrome, aura)-	59 %
2.	Able to tell that a migraine is coming through visual changes or distortions-	43 %
3.	The head pain frequently exists on one side or the head only-	91 %
4.	The head pain usually exists in the temporal regions (eye level at side of head)-	70 %
5.	The head pain usually exists in the forehead region between the eyebrows and the hairline-	43 %
6.	The head pain usually begins in the neck at the base of the head and then radiates towards the temporal and forehead regions-	39 %
7.	The headache occurs in many different regions of the head from time to time-	30 %
8.	The head pain usually occurs in the region at the top of the head-	22 %
9.	Frequently the headaches are throbbing, pulsating headaches-	87 %
10.	The headaches are usually characterized by pressure on the head, the sensation of which might be described as a tight band across the forehead and around the head-	52 %
11.	The headaches usually occur only during menses-	9 %
12.	The headaches occur during menses and at many other times-	78 %
13.	Nausea or vomiting generally accompany the headaches-	80 %
14.	Sensitivity to light generally accompanies the headaches-	94 %

. . . continued

	Percentage of Respondents--
15. Sensitivity to sound generally accompanies the headaches-	96 %
16. Tears and nasal stuffiness generally accompanies the headaches-	35 %

When experiencing a stressful situation the following reactions are experienced-

17. Oily skin	9 %
18. Sweaty feet	30 %
19. Flushed face	80 %
20. Frequent need to urinate	54 %
21. Cold hands	61 %
22. Burping	0 %
23. Face feels hot	87 %
24. Tight stomach muscles	69 %
25. Sweaty hands	70 %
26. Gasiness	22 %
27. Acid stomach	31 %
28. Shallow, rapid breathing	46 %
29. Cold feet	46 %
30. Diarrhea	19 %
31. Palpitation	57 %
32. Short breath	44 %
33. shaky hands	59 %

N=54

APPENDIX N

ANALYSIS OF VARIANCE SOURCE TABLES

Summary of Analysis of Variance for Four Treatment Groups

Across Five Repeated Measures

Headache Days

Source of Variation	SS	df	MS	F	P
Between Subjects	3216.04	47			
'A' Main Effects	340.51	3	113.50	1.74	0.173
Subjects Within Gps	2871.00	44	65.25		
Within Subjects	1262.00	192			
'B' Main Effects	413.89	4	103.47	21.92	0.001
'A X B' Interaction	27.30	12	2.28	0.48	0.923
'B' X Subj Within Gps	830.75	176	4.72		

Summary of Analysis of Variance for Four Treatment Groups

Across Five Repeated Measures

Headache Hours

Source of Variation	SS	df	MS	F	P
Between Subjects	695918.25	47			
'A' Main Effects	85646.50	3	28548.85	2.04	0.122
Subjects Within Gps	616376.56	44	14008.56		
Within Subjects	253645.00	192			
'B' Main Effects	67140.06	4	16785.02	16.33	0.001
'A X B' Interaction	8155.39	12	679.62	0.66	0.787
'B' X Subj Within Gps	180888.69	176	1027.78		

Summary of Analysis of Variance for Four Treatment Groups

Across Five Repeated Measures

Headache Index

Source of Variation	SS	df	MS	F	P
Between Subjects	16479.57	47			
'A' Main Effects	1757.76	3	583.92	1.74	0.174
Subjects Within Gps	14802.66	44	336.42		
Within Subjects	6327.40	192			
'B' Main Effects	554.54	4	138.64	4.32	0.002
'A X B' Interaction	118.36	12	9.86	0.31	0.988
'B' X Subj Within Gps	5648.07	176	32.09		

Summary of Analysis of Variance for Two Responsivity Groups

Across Five Repeated Measures

Headache Days

Source of Variation	SS	df	MS	F	P
Between Subjects	3216.04	47			
'A' Main Effects	11.05	1	11.05	0.16	0.692
Subjects Within Gps	3204.99	46	69.67		
Within Subjects	1262.00	192			
'B' Main Effects	403.36	4	100.84	21.87	0.001
'A X B' Interaction	10.16	4	2.54	0.55	0.699
'B' X Subj Within Gps	848.48	184	4.61		

Summary of Analysis of Variance for Two Responsivity Groups

Across Five Repeated Measures

Headache Hours

Source of Variation	SS	df	MS	F	P
Between Subjects	695918.25	47			
'A' Main Effects	11787.75	1	11787.75	0.79	0.378
Subjects Within Gps	684130.44	46	14872.40		
Within Subjects	253645.00	192			
'B' Main Effects	64908.04	4	16227.01	15.99	0.001
'A X B' Interaction	2038.50	4	509.62	0.50	0.734
'B' X Subj Within Gps	186699.62	184	1014.67		

Summary of Analysis of Variance for Two Responsivity Groups

Across Five Repeated Measures

Headache Index

Source of Variation	SS	df	MS	F	P
Between Subjects	16479.57	47			
'A' Main Effects	56.55	1	56.55	0.16	0.692
Subjects Within Gps	16423.02	46	357.82		
Within Subjects	6327.40	192			
'B' Main Effects	569.06	4	142.26	4.59	0.001
'A X B' Interaction	58.41	4	14.60	0.47	0.757
'B' X Subj Within Gps	5699.96	184	30.98		

Summary of Analysis of Variance for Two Treatment Groups

Across Five Repeated Measures

Headache Days

Source of Variation	SS	df	MS	F	P
Between Subjects	3216.04	47			
'A' Main Effects	0.08	1	0.082	0.001	0.973
Subjects Within Gps	3215.96	46	69.91		
Within Subjects	1262.00	192			
'B' Main Effects	402.73	4	100.68	21.67	0.001
'A X B' Interaction	3.62	4	0.90	0.20	0.941
'B' X Subj Within Gps	855.03	184	4.65		

Summary of Analysis of Variance for Two Treatment Groups

Across Five Repeated Measures

Headache Hours

Source of Variation	SS	df	MS	F	P
Between Subjects	695918.25	47			
'A' Main Effects	9389.42	1	9389.42	0.63	0.432
Subjects Within Gps	686528.62	46	14924.54		
Within Subjects	253646.00	192			
'B' Main Effects	64845.97	4	16211.50	15.97	0.001
'A X B' Interaction	1952.60	4	488.15	0.48	0.750
'B' X Subj Within Gps	186787.31	184	1015.15		

Summary of Analysis of Variance for Two Treatment Groups

Across Five Repeated Measures

Headache Index

Source of Variation	SS	df	MS	F	P
Between Subjects	16479.57	47			
'A' Main Effects	192.74	1	192.74	0.54	0.464
Subjects Within Gps	16286.82	46	354.06		
Within Subjects	6327.40	192			
'B' Main Effects	561.90	4	140.47	4.52	0.002
'A X B' Interaction	38.11	4	9.53	0.31	0.873
'B' X Subj Within Gps	5720.25	184	31.09		

Summary of Analysis of Variance for Two Groups*

Across Five Repeated Measures

Headache Days

Source of Variation	SS	df	MS	F	P
Between Subjects	3216.04	47			
'A' Main Effects	338.25	1	338.25	5.41	0.025
Subjects Within Gps	2877.79	46	62.56		
Within Subjects	1262.00	192			
'B' Main Effects	415.68	4	103.92	22.66	0.001
'A X B' Interaction	14.68	4	3.67	0.80	0.527
'B' X Subj Within Gps	843.97	184	4.59		

* Group 1: EMG recoverers/EMG training
ST recoverers/ST training

Group 2: EMG recoverers/ST training
ST recoverers/EMG training

Summary of Analysis of Variance for Two Groups*

Across Five Repeated Measures

Headache Hours

Source of Variation	SS	df	MS	F	P
Between Subjects	695918.25	47			
'A' Main Effects	52583.99	1	52583.59	3.76	0.059
Subjects Within Gps	643334.25	46	13985.52		
Within Subjects	253646.00	192			
'B' Main Effects	67361.38	4	16840.34	16.73	0.001
'A X B' Interaction	3555.84	4	888.96	0.88	0.475
'B' X Subj Within Gps	185183.44	184	1006.43		

* Group 1: EMG recoverers/EMG training
ST recoverers/ST training

Group 2: EMG recoverers/ST training
ST recoverers/EMG training

Summary of Analysis of Variance for Two Groups*

Across Five Repeated Measures

Headache Index

Source of Variation	SS	df	MS	F	P
Between Subjects	16479.57	47			
'A' Main Effects	1359.30	1	1359.30	4.14	0.048
Subjects Within Gps	15120.28	46	328.70		
Within Subjects	6327.40	192			
'B' Main Effects	565.25	4	141.31	4.52	0.002
'A X B' Interaction	5.88	4	1.47	0.05	0.996
'B' X Subj Within Gps	5752.48	184	31.26		

* Group 1: EMG recoverers/EMG training
ST recoverers/ST training

Group 2: EMG recoverers/ST training
ST recoverers/EMG training

Summary of Analysis of Variance for Two Groups--Criterion Grouping Across Five Repeated Measures

Headache Days

Source of Variation	SS	df	MS	F	P
Between Subjects	3216.04	47			
'A' Main Effects	57.76	1	57.76	0.841	0.364
Subjects Within Gps	3158.29	46	68.66		
Within Subjects	1262.00	192			
'B' Main Effects	296.69	4	74.17	16.67	0.000
'A X B' Interaction	39.78	4	9.94	2.24	0.067
'B' X Subj Within Gps	818.86	184			

Summary of Analysis of Variance for Two Groups--Criterion Grouping Across Five Repeated Measures

Headache Hours

Source of Variation	SS	df	MS	F	P
Between Subjects	695918.25	47			
'A' Main Effects	13530.66	1	13530.66	.0.91	0.345
Subjects Within Gps	682387.25	46	14834.50		
Within Subjects	253645.00	192			
'B' Main Effects	50947.32	4	12736.83	12.80	0.000
'A X B' Interaction	5711.50	4	1427.87	1.44	0.224
'B' X Subj Within Gps	183026.88	184	994.71		

Summary of Analysis of Variance for Two Groups--Criterion Grouping Across Five Repeated Measures

Headache Index

Source of Variation	SS	df	MS	F	P
Between Subjects	16479.57	47			
'A' Main Effects	88.40	1	88.40	0.25	0.621
Subjects Within Gps	16391.17	46	356.33		
Within Subjects	6327.40	192			
'B' Main Effects	379.67	4	94.92	3.15	0.016
'A X B' Interaction	207.54	4	51.88	1.72	0.147
'B' X Subj Within Gps	5550.80	184	30.17		

Summary of Analysis of Variance for Weaned, Not-Weaned Groups

Across Five Repeated Measures

Headache Days

Source of Variation	SS	df	MS	F	P
Between Subjects	3216.04	47			
'A' Main Effects	72.71	1	72.71	1.06	0.308
Subjects Within Gps	3143.34	46	68.33		
Within Subjects	1262.00	192			
'B' Main Effects	454.13	4	113.53	27.06	0.001
'A X B' Interaction	86.77	4	21.69	5.17	0.001
'B' X Subj Within Gps	771.87	184	4.20		

Summary of Analysis of Variance for Weaned, Not-Weaned Groups

Across Five Repeated Measures

Headache Hours

Source of Variation	SS	df	MS	F	P
Between Subjects	695918.25	47			
'A' Main Effects	18819.07	1	18819.07	1.28	0.264
Subjects Within Gps	677099.19	46	14719.55		
Within Subjects	253645.00	192			
'B' Main Effects	71931.12	4	17982.78	19.19	0.001
'A X B' Interaction	16343.82	4	4085.96	4.36	0.002
'B' X Subj Within Gps	172394.38	184	936.93		

Summary of Analysis of Variance for Weaned, Not-Weaned Groups

Across Five Repeated Measures

Headache Index

Source of Variation	SS	df	MS	F	P
Between Subjects	16479.57	47			
'A' Main Effects	759.31	1	759.31	2.22	0.143
Subjects Within Gps	15720.27	46	341.74		
Within Subjects	6327.40	192			
'B' Main Effects	729.90	4	182.47	6.52	0.000
'A X B' Interaction	609.17	4	152.29	5.44	0.000
'B' X Subj Within Gps	5149.15	184	27.98		